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THE PATHOLOGY OF TRANSFUSION REACTIONS*

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NEW ORLEANS

INTRODUCTION

The text of this presentation will deal only with the hemolytic transfusion reaction caused by accidental administration of incorrect blood types and Rh factors. The hemolytic type of reaction is frequently fatal. An understanding of the pathologic anatomy and the disturbed physiology of this condition is essential to proper treatment. Although much light has been shed on this subject within recent years, there still remain many unanswered questions. Anyone who has had the unfortunate experience of encountering a fatal transfusion reaction realizes more than ever how perplexing this problem can become.

It is a well recognized fact that the pathology of hemolytic transfusion reaction is very similar to, and objectively the same as in many other conditions. The crush syndrome, shock, mushroom poisoning, burns, heat stroke, sulfonamide intoxication, and toxemias of pregnancy may produce the same pathologic findings. The pathologic anatomy primarily involves the kidney in these conditions and, of course, there is other pathology secondary to renal failure.

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The renal pathology has most often been described in the literature under the heading of "Lower Nephron Nephrosis" or "Hemoglobinuric Nephrosis". An exhaustive study of this subject has been recently reported by Lucke[1].

GROSS PATHOLOGY

The kidneys are usually swollen and edematous in appearance; their weight is from slightly to much increased. There is a tendency for greater swelling when the survival period is longer.[1] The capsule strips with ease from a previously normal kidney showing a smooth pale outer surface. When the organ is sectioned, the pale cortex bulges, is wider than normal and is sharply demarcated from the brownish purple well striated medullary portion. Occasionally small hemorrhages may be noted in the calices and pelvis.

MICROSCOPIC PATHOLOGY

There is some divergence of opinion regarding the histopathology. According to Lucké[1], Mallory[2] and others, the main objective histopathologic features are found in the lower segment of the nephron where degeneration or actual necrosis involves selectively focal portions of the thick tubules of Henle and the distal convoluted tubules. The more severely disintegrated portions of the tubules are apt to provoke edema and cellular reaction in the surrounding stroma, and thrombosis of adjacent veins may occur. Brownish or eosinophilic granular casts are found in the distal segments and collecting tubules. Although focal degeneration of epithelium has been noted in the collecting tubules, such changes are less fre-

quent and nearly always adjacent to heme casts. Frequently the proximal convoluted tubules appear dilated and their lining cells swollen. Of this latter point, there can be little doubt, but Mallory[2] points out, frozen sections on formalin fixed tissue suggest that the appearance of dilatation is largely, but not entirely artefact due to shrinkage. Moon[3] states that in general all portions of the convoluted tubules are affected and believes the more pronounced changes to be in the upper segment. This view is at variance with that of most other observers, although minimal objective changes, i. e., slight "cloudy swelling" and occasional fatty deposits have been noted. According to Lucké actual necrosis in the proximal segment is very rare. The glomeruli are usually normal in size and cellularity but with poor blood content, suggesting inadequate circulation. Moon,[3] however, is impressed with the hyperemia of the glomerular tufts. Recent work by Trueta[4] *et al* and others would seem to support glomerular ischemia, except in the juxtamedullary glomeruli. There is frequently an eosin-staining, granular or globular precipitate in the subcapsular spaces, indicating an increased glomerular permeability. At times, there is some swelling of the normally flat epithelium of the capsule, especially near the mouth.

Time permits only a summary of the detailed pathology as follows: The damage to the tubular cells may vary from very slight to complete necrosis, at times even with rupture of tubular walls and extrusion of casts into the interstices. When this occurs, varying amounts of reactionary elements collect about the cast forming a "granuloma" which in time undergoes hyalin change. The extruded cast is usually hyalin in nature rather than one of the more numerous heme casts. Regeneration of tubular epithelium starts after three or four days' survival and is almost complete if the patient survives for ten days or more. Ayer and Gould[5] have described autopsy findings in 7 patients who died three hours to ten days after hemolytic reactions.

PATHOGENESIS AND DISTURBED PHYSIOLOGY

It seems fairly well accepted that no one single factor can account for the severe renal anatomic and functional pathology in this condition. That precipitated hemoglobin causes sufficient mechanical blockage to cause severe anuria is doubtful. Many tubules are not involved with casts. Yuile *et al*[6] and others have been unable to produce renal insufficiency in dogs with single massive intravenous doses of hemoglobin regardless of the reaction of urine. While it is true that epithelial degeneration is sometimes noted adjacent to acid hematin casts, it has not been conclusively proved that this substance alone is definitely toxic. March hemoglobinuria and paroxysmal hemoglobinuria do not ordinarily produce renal insufficiency. Human experiments support the hypothesis that hemoglobin is excreted in large quantities only when the permeability of the glomerular filters is increased and that albuminuria coincidentally appears in all cases.[7] This increased permeability could be explained on oxygen deficiency due to ischemia.

Mallory[2] suggests the possibility that anaphylaxis contributes to the reaction to incompatible blood. Certainly in severe reactions a state of shock is not unusual.

The hypothesis that disturbed renal blood flow plays a major role in this condition is receiving much attention and recently much factual data have been published in regard to cortical ischemia and renal medullary shunt mechanism. Trueta[4] *et al* have made important studies concerning renal pathology caused by neurovascular disturbances. They have shown very convincingly that with appropriate nerve stimulation, both in animals with the abdomen unopened and in those in which the kidneys have been exposed, the renal blood flow may be diverted from its commonly accepted course, and that as a result the cortex may be partly or wholly deprived of its blood supply. These studies were carried out with the use of radio-opaque and dye substances. They found that the renal circulation time was shortened by one-half, though the calibre of the renal artery at the same time was re-

duced by one-quarter. This is strong evidence that a vascular short-circuiting mechanism exists. That the blood is being diverted from the cortex and short-circuited through medullary (especially subcortical) blood channels is proved by radiographic and other evidence. Further experimental work by the same group[11] shows that the blood is shunted through the juxtamedullary glomeruli to the vasa recta components and back to the interlobular veins. The outer cortex therefore remains relatively ischemic to varying degrees, while the inner cortex and medulla are hyperemic. They therefore show that in the experimental animal a renal circulation can be continued through the medulla while most of the cortex is functionally ischemic. They note also that under these conditions the flow of urine in the ureter decreases or is entirely suppressed. They suggest, with much reason, that nerve stimulation can be produced centrally or peripherally by a variety of noxious agents and that the picture seen in many loosely related syndromes —e.g., "sulfa kidney", incompatible-transfusion kidney, etc., is a result of a defense device by which the cortex of the kidney is excluded from the circulating toxin or other noxious agent and thus protected. Too prolonged operation of the device results in permanent damage. Only recently I have had the opportunity to study necropsy material on the kidneys of a fourteen year old girl with bilateral coalescent cortical infarctions following furunculosis of the lower extremities. It is interesting speculation and quite possible that protective mechanisms against bacterial toxins may have caused this picture. No other obvious cause could be found in this instance. The picture is similar to the cortical necrosis that has been associated with postabortum state. The gross and microscopic picture presented is identical to the experimental lesions produced by Trueta[4] *et al.*

Corcoran and Page[8] point out that the principal lesion resulting from complete obliteration of renal arterial inflow is degeneration of the proximal convoluted tubules and suggest that this picture is different from that of lower nephron nephrosis. It would seem logical to assume that complete renal ischemia should produce a different picture from that of incomplete ischemia or medullary "short circuit" as described by Trueta[4] *et al* for the following reasons: In the first instance, the blood supply is completely cut off from the glomerular apparatus as well as all tubules. Complete anuria must exist. In the latter instance, cortical ischemia is a relative thing and may be of varying degrees of intensity. It must be assumed that unless complete anuria exists that some blood must pass through the afferent arterioles into the glomerulus. This amount may be and undoubtedly is often very small and of low hydrostatic pressure. It may be sufficient however to prevent complete anoxia of the glomeruli and to prevent marked objective histopathologic changes but not yet enough to prevent altered physiology, i.e., increased capillary permeability. Since practically all of the arterial blood of the kidney passes through the glomeruli before being distributed to the rest of the organ (Maximow and Bloom[9]) it can be assumed that the glomeruli would have first call on whatever small amount of blood-borne oxygen that may later reach the renal tubules. Both the proximal and the distal convoluted tubules then have reason to show more objective pathology than the glomeruli. As has been pointed out, there is difference of opinion as to which set of convoluted tubules show the most change. These differences may possibly be explained on a basis of priority of blood supply from the efferent arteriole of the glomerulus. Since the minute circulation of the cortex is still the subject of investigation, this point is open to question.

MECHANISM OF OLIGURIA

From reviewing the physiologic and pathologic mechanisms discussed it appears that several factors must be considered in the mechanism of oliguria.

The hypothesis that the tubules are so completely obstructed by heme pigment

as to cause oliguria is open to serious question, for in many instances insufficient tubules appear obstructed. If mechanical obstruction were the sole cause, one would expect a severe grade of internal hydronephrosis with dilatation of tubules above the point of obstruction and dilatation of the space between Bowman's capsule and the glomerular tufts. Such is not the case.

In the light of present knowledge, it seems quite likely that altered renal blood flow plays the fundamental role in production of the pathologic, anatomic, and physiologic findings. It is well accepted that renal blood flow may be tremendously decreased in shock states, sometimes as much as one-twentieth of normal values.[10] Experimental procedures mentioned previously have proved that in certain conditions, the cortical blood supply may be almost entirely shunted to the medulla.[4] Changes due to anoxia necessarily follow, producing altered physiology and degenerative changes in the glomeruli and tubules as described. Further degenerative disease may possibly be caused by the presence of heme products. It may well be then that the oliguria is brought about as follows: (1) Degenerative and physiologic changes are caused by altered blood flow and cortical ischemia. (2) Decreased hydrostatic pressure in the glomeruli produces less filtrate. (3) Such filtrate as passes the glomeruli may be taken back into the circulation by unselective resorption of the damaged tubules. (4) The possible factors of mechanical obstruction due to heme casts, interstitial reaction, and edema must be taken into consideration.

SUMMARY

1. The altered physiology and pathologic findings in hemolytic transfusion reactions are similar to those of many other conditions producing the picture of "lower nephron nephrosis".

2. Recent experimental data strongly suggest that the pathology and physiologic changes are not confined to the lower nephron, but include the entire nephron, and are comparable with the changes found in ischemic cortical necrosis previously described associated with postabortive cases.

REFERENCES

1. Lucké, Baldwin: Lower nephron nephrosis. Mil. Surg., 99:371-396, 1946.
2. Mallory, T. B.: Hemoglobinuric nephrosis in traumatic shock. Am. J. Clin. Path., 17:427-443, 1947.
3. Moon, Virgil H.: Renal deficiency. J. A. M. A., 134:425-429, 1947.
4. Trueta, J., Barclay, A. E., Daniel, P., Franklin, K. J., and Pritchard, M. M. L.: Renal pathology in the light of recent neurovascular studies. Lancet, 2:239, 1946.
5. Ayer, G. D., and Gould, A. G.: Uremia following blood transfusion. Nature and significance of renal changes. Arch. Path., 33:513-532, 1942.
6. Yuile, C. L., Gold, M. A., and Hinds, E. G.: Hemoglobin precipitation in renal tubules: study of its causes and effects. J. Exper Med., 82:361-374, 1945.
7. Gilligan, D. R., Altschule, M. D., and Katersky, E. M.: Studies of hemoglobinemia and hemoglobinuria produced in man by intravenous injection of hemoglobin solutions. J. Clin. Investigation, 20:177-187, 1941.
8. Corcoran, A. C., and Page, Irvine H.: Crush syndrome. J. A. M. A., 134:436-441, 1947.
9. Maximow, A. A., and Bloom, Wm.: A Textbook of Histology. W. B. Saunders Company, 493-494, 1942
10. Richards, D. W., Jr.: Circulation in Traumatic Shock in Man. Harvey lecture, Feb. 17, 1944.
11. Trueta, J., Barclay, A. E., Daniel, P. M., Franklin, K. J., and Pritchard, M. M. L.: Studies of the Renal Circulation. Charles C. Thomas Publisher, 1947.

CAUSES AND PREVENTION OF TRANSFUSION REACTIONS*

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NEW ORLEANS

Attempts to classify transfusion reactions seem inevitably to run afoul of the old chicken-and-egg or "which came first" fable. Some causes are well understood, while their effects, as exhibited by the patient, are in many cases but imperfectly known. Conversely, rather well defined reactions are encountered in which the true nature of the etiologic agents remains obscure. The causes of some reactions are entirely exogenous. Others may be endogenous, while in many both exogenous and endogenous factors are concerned.

NON-HEMOLYTIC REACTIONS

Pyrogenic Reactions: Pyrogen may be loosely defined as any substance which when injected intravenously will cause an elevation of temperature. This would in-

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clude all sorts of debris remaining in improperly prepared blood-collecting or administering equipment: old blood clots, dried proteins, bacteria, lint, etc. More strictly, pyrogens are water soluble, toxic products of bacterial metabolism and are extremely thermostable and capable of passing through very fine bacterial filters.

Pyrogenic reactions usually manifest themselves within ten to thirty minutes following the start of the transfusion:

A. Mild form:

1. Chilly sensations or mild rigor.
2. Relatively little temperature increase (103° F. maximum).
3. Slight frontal headache at times.
4. Some apprehension.
5. Temperature subsides in an hour or so.

B. More severe form:

1. Moderate to marked rigors.
2. Temperature may reach 105° F.
3. Flushing of facies.
4. Post-sternal oppression, with or without dyspnea and cyanosis.
5. Frontal headache.
6. Muscular pains.
7. Nauseated in some instances.
8. Apprehension usually acute.
9. Fever may persist for four to six hours.

Not all of the above signs or symptoms are necessarily exhibited by any one patient. I have never personally encountered a fatal pyrogenic reaction.

Prevention of pyrogenic reactions hinges on keeping pyrogen out of all apparatus used for collecting, transferring or administering blood. This simply means thorough and scrupulous cleansing and sterilization of all apparatus. I would stress these basic principles:

1. Flushing of all apparatus with tap water *immediately after use.*
2. Keeping all apparatus immersed in water until ready to re-prepare.
3. High pressure tap water flushing of rubber tubing for minimum of two hours, each length of tubing to be vigorously stretched throughout its length several times during this phase.
4. Thorough soaking of all glass, metal and non-tubular rubber parts in a detergent for twenty-four hours.
5. Complete rinsing to remove all detergent or soap used in step 4.
6. Flushing of all parts of each set with copious quantities of *fresh, pyrogen-free* distilled water immediately before assembly.
7. Sterilization of apparatus *within one hour of assembly.*
8. Operation of autoclave by time and temperature method only.
9. The assignment of competent nonrotating personnel to all the above duties.

Time does not permit a detailed discussion of the principles of *water-still* construction and operation. The type of still required will be largely determined by the size of the hospital and the nature of the raw water supply. I would personally recommend the Barnstead Type "Q" still as minimum standard. In some localities double or triple stills should be used.

Pyrogen-free distilled water is obtainable commercially in hermetically sealed flasks, but is too expensive except for emergency or small scale operations. Such water will *not* remain pyrogen free indefinitely once the hermetic seal is broken.

Until recent years it was customary to use stainless-steel mesh filters in administering citrated blood. These require cleansing in C. P. nitric acid and we discontinued them about eighteen months ago in favor of disposable plastic filter elements.

Many small and some large hospitals now use the efficient, disposable collecting and transfusing sets provided by the two major commercial solution laboratories for use with their standard vacuum transfusion bottles.

A container of blood should not be opened until one is actually ready to administer it to the patient. If, for any reason, the blood is opened but not used, the top should be promptly covered with several layers of sterile gauze and the bottle returned at once to the laboratory refrigerator. We feel that

blood so opened should not be used after six hours.

Thermal Reactions: This is a classification developed in an effort to remove some of the obscure non-hemolytic, *pyrogen-like* reactions in which the following circumstances seem to be involved:

1. The condition of the patient. In a toxic individual exhibiting a spiking temperature curve, the administration of blood sometimes gives rise to chill and febrile reactions. In view of the complex nature of whole blood and the altered blood chemistry of such patients, we believe that many apparently mild pyrogenic reactions experienced by them actually are coincidental and therefore not true untoward transfusion reactions.

2. The temperature of the blood. Since, with rare exceptions, we use only refrigerated bank blood on our transfusion service, we have at times felt that mild chills, especially in toxic or debilitated patients, may be actually due to the administration of cold blood, especially where 500 c.c. have been administered in sixty to seventy-five minutes.

The heating of blood shoud be forbidden, unless it is done in a properly controlled incubator under competent supervision. (Maximum temperature not to exceed 99° F.) Excessive heating of blood or plasma can alter the proteins sufficiently to produce severe reactions. For the great majority of patients I see no need for any warming of the blood.

Allergic Reactions: In our experience this has been the most common type of non-hemolytic reaction. At present it is believed that allergic antigens and/or antibodies in the donor's blood are responsible.

Mildest and most frequent allergic reaction is simple urticaria, appearing after administration of from 50 to 500 c.c. of blood. At times slight angioneurotic edema appears. Eosinophilia is not uncommon, as is mild fever. Itching may precede appearance of wheals.

More severe allergic reactions are denoted by asthma, with or without the above signs. Infrequently the asthmatic seizure is severe and acute laryngeal edema and bronchospasm develop, while involuntary passage of the excreta may occur.

The most severe form of allergic reaction may begin with urticaria and angioneurotic edema followed rapidly by the development of anaphylactoid shock with marked hypotension. Very rarely death will ensue.

At present, prevention of allergic reactions rests chiefly on careful selection and control of donors:

1. Donors are requested to refrain from all foods and beverages for a minimum of four hours before phlebotomy, with the following exceptions: dry toast and jelly, plain coffee or tea with sugar, fruits, fruit juices.

2. No person is accepted as a donor who gives a history of urticaria, asthma or other allergy within one year.

The patient, especially if presenting a history of allergy, may be given 50 mgm. of Benadryl orally about two hours before starting the transfusion, at which time the dose may be repeated. Or a single 50 mgm. dose may be given intravenously just prior to beginning the transfusion. Proper cleansing of apparatus contributes to prevention of allergic reactions.

Psychogenic Reactions: Undoubtedly the patient's state of mind at times is sufficient to produce mild chilly sensations and even low grade fever. Such a situation can be confused with a pyrogenic reaction. Further, psychogenic and neurogenic factors undoubtedly are concerned in allergic reactions.

Reactions Due to Physical Factors: Cardiac and/or vascular mishaps, while extremely rare, as results of transfusion, can occur in the following circumstances:

1. Overloading of the circulation by too rapid administration of blood, especially in large volume. This is a real danger when the heart is partially decompensated. Transfusion is, of course, contraindicated in severe pulmonary edema.

2. Embolism will occur if small clots are permitted to enter the patient's circulation.

This is unlikely to happen if a properly assembled *filter* dripmeter is used. Forced administration of blood by applying positive pressure to the bottle is potentially dangerous in that it may cause a break in the filter. Further, it *may* allow administration of air sufficient to cause serious or fatal embolism.

HEMOLYTIC TRANSFUSION REACTIONS

All hemolytic reactions due to transfusion of whole blood or red cells have a common serologic basis: incompatibility between bloods of donor and recipient. Typically, the donor's erythrocytes possess antigens (agglutinogens) absent from the red cells of the recipient, while the latter's serum contains one or more antibodies specific for the donor's agglutinogens.

Some antibodies exist naturally and are known as normal antibodies. Best known examples are the anti-A and anti-B isohemagglutinins. These are specific for the A and B factors, which determine the four major blood groups: A, B, AB, O, of Landsteiner.

In addition to the normal isoantibodies there are the *irregular* or *isoimmune* antibodies. These result from immunization of the individual by antigens which have entered the blood stream by transfusion and/or pregnancy, the recipient lacking such antigens in his or her erythrocytes, thereby filling one requirement for isoimmunization.

Most frequently encountered irregular antibodies are those caused by immunization to one or more of the components of the Rh complex. Only rarely have persons been found who exhibited antibodies specific for the P, M or N factors. Rh isoimmunization, however, is known to be responsible for over 90 per cent of intragroup hemolytic transfusion reactions. Thus in preparing blood for transfusion, one must be constantly aware of the necessity of achieving both group and Rh compatibility. The latter is especially important in female patients for these reasons:

1. Patient may be Rh negative and actually possess Rh antibodies derived from pregnancy.

2. Transfusion of Rh positive blood into any Rh negative female may initiate an immunization which might result in an erythroblastotic baby years hence.

Rh negative male patients known or believed to be Rh immunized, should, of course, receive only compatible Rh negative blood in order to prevent the possibility of a hemolytic transfusion reaction.

The onset of a hemolytic reaction is presumably characterized by agglutination, but hemolysis of the donor cells quickly moves into the picture, as evidenced by the appearance of hemoglobinemia and hemoglobinuria within a few hours following transfusion of 50 c.c. or more of incompatible blood. The severity of hemolytic reactions is affected by a number of variables such as:

1. Quantity of blood transfused.

2. Strength and specificity of recipient's antibodies.

3. Rate of transfusion and rate of intravascular hemolysis.

4. General physical status of the recipient.

Perhaps the most prominent symptom of a hemolytic transfusion reaction is pain in the lumbar region. This tends to occur immediately and is usually accompanied or followed by dyspnea, cyanosis, and a sense of thoracic constriction. Nausea, vomiting, and abdominal pain are not uncommon occurrences and chills and fever may usher in the reaction. The patient is usually quite apprehensive. Hypotension is common with pulse rapid and weak. These signs and symptoms, in various combinations, may be said to represent the acute phase of the reaction. (Instantaneous death due to cardiac or cerebral embolism by masses of agglutinated donor cells is a possibility but is so rare as to be almost unknown.)

Usually the acute phase subsides in several hours and the patient rests fairly easy. Jaundice will usually appear in eighteen to thirty-six hours. Some patients show little

if any decrease in the renal function while others exhibit urinary suppression for twenty-four hours to fourteen days. The uremic state intervenes and may terminate in death, although some patients will gradually regain their kidney function and survive. Although hemolytic reactions are rare, the death rate has ranged from 50 to 90 per cent for many years.

In the event a hemolytic reaction is suspected, one should immediately obtain blood and urine samples to inspect for free hemoglobin. Blood groups and Rh types of patient and donor should be rechecked, as well as the cross-matching.

The present discussion does not consider the treatment of reactions, but, for most of them, prevention is by far the best therapy. This is fearfully true of the hemolytic variety. Observance of a few essential safety rules will do much to avert such tragic episodes:

1. Accurate and complete labeling of all blood samples and bottles of blood.

2. The use of potent, *human* grouping and Rh typing sera.

3. Careful cross-matching of patient's and donor's bloods.

4. Close observation of the patient during administration of the first 50-100 c.c. of blood, with immediate cessation if symptoms or signs of hemolytic reaction arise. Patients under general anesthetic must be constantly observed. Potential danger here is great.

Be sure before transfusing. Recheck all procedures employed in grouping, Rh typing, cross-matching and identifying of all bloods concerned. It has been said that a bottle of blood is like a stick of dynamite. Both are capable of causing great destruction if misused.

ANTICOAGULANTS IN THE TREATMENT OF POSTPARTUM VENOUS THROMBOSIS AND EMBOLISM*

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The past decade has seen the physician's armamentarium for the treatment of thrombo-embolic phenomena enhanced by the addition and clinical application of two potent anticoagulant substances; namely, heparin and dicumarol. While a considerable literature has developed relating to the clinical experiences with these preparations in the treatment of medical and postoperative conditions, we were able to discover but one report, that of Davis and Porter,[1] relating to their use in any series of patients in the postpartum state. Therefore, without entering into a discussion as to the relative merits of this treatment and treatment by means of sympathetic block and ligation of the superficial femoral veins, since we have had practically no experience with the latter method of treatment, we are reporting our experiences with the use of heparin and dicumarol in obstetric patients.

GENERAL CONSIDERATIONS

Heparin.—Heparin was discovered some thirty years ago by Howell.[2] It is thought to be produced in the body by the mast cells of Ehrlich. It is a mucoitin polysulfuric acid and its activity is presumed to be owing to its strong negative electric charge. The effect of heparin on the blood is instantly neutralized by the addition of basic protamine. Soon after heparin was obtained in pure state in 1935, it was utilized in the treatment of thrombosis, and Murray and Best[3] and Murray[4] in Toronto

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reported the successful use of the drug in thrombophlebitis and embolism. The characteristic almost instantaneous action of this substance makes it desirable when a rapid response is indicated. The use of heparin does not require the meticulous laboratory control which is necessary for the safe use of the other anticoagulant, since, should bleeding occur, the drug can be withheld and ordinarily the blood can be expected to return to its normal state of coagulability within a period of a half to two and a half hours. However, the dosage ordinarily is determined on the basis of the coagulation time of the blood. An effort is made to maintain the clotting time between twenty and twenty-five minutes.

The disadvantages of the use of heparin are that it must be administered parenterally and that it is relatively expensive, both facts mitigating against prolonged use of the drug in any given case.

Dicumarol.—Roderick[5] first recognized that hemorrhagic sweet clover disease of cattle was due to a product which resulted from the spoilage of clover hay and which impaired the coagulation of blood. The fascinating story by Link,[6] of the University of Wisconsin, given in the Harvey Lectures in 1943 and 1944, details the isolation, identification, and later the synthesis of this compound by him and his co-workers and describes, indeed, a masterpiece of scientific endeavor.

Dicumarol, which chemically is 3,3′ methylenebis (4-hydroxycoumarin), is a crystalline substance with a melting point of 288° to 289° F., which, when administered orally to animals and human beings, so impairs the ability of the liver to produce prothrombin that the resultant prothrombin deficiency vitiates the coagulation of blood. In toxic doses this substance produces central necrosis of the hepatic lobule, and death from generalized hemorrhage occurs. As a secondary effect of prothrombin deficiency the coagulation time of the blood may be prolonged, but this effect does not necessarily parallel the degree of prothrombin deficiency and hence the coagulation time cannot be used safely as the basis for controlling administration of dicumarol.

The effect of dicumarol is relatively slow in appearing. An interval of twenty-four to forty-eight hours elapses from the time the substance is administered until its full effect is meted in terms of prothrombin deficiency, but the effect is maintained several days after the drug has been withdrawn. That the effects on the liver, when dicumarol is administered in therapeutic doses, are not permanent is displayed by the fact that liver function tests give normal results even after this prophylactic agent has been administered for several months.

As in animals, so in human beings there has been shown to be considerable individual variance in tolerance to the drug, and Link[6] showed that in animals this variation in tolerance was an inherited trait, passing according to the Mendelian law. The abovementioned characteristics make it imperative that dicumarol should never be given to a patient unless the dosage can be gauged on the basis of the daily determination of blood prothrombin time, lest hazardous results supervene.

Dosage.—For the purpose of clarifying the data on dosage which are to follow, a word of explanation regarding the determination of the prothrombin time is given.

At the laboratory in the Mayo Clinic a Magath modification of the Quick method is used, giving a normal prothrombin time of 17 to 19 seconds, which may be considered 100 per cent. A prothrombin time of 27 seconds signifies that the value for prothrombin is 30 per cent of normal, one of 35 seconds signifies a value of 20 per cent of normal and one of 58 seconds signifies a value of 10 per cent of normal.

It is to be pointed out that these values will vary for different laboratories in which different technics and different thromboplastins are used. The physician who is to supervise dicumarol therapy will find it necessary, therefore, to familiarize himself with the relationship of prothrombin time to prothrombin percentage as determined in his particular laboratory. Allen and Barker and their associates[7] at the

clinic, in a clinical study of more than 2,300 medical and surgical patients who received dicumarol, have shown that serious bleeding will almost never occur if the value for prothrombin is greater than 10 per cent of normal, and that, on the other hand, thrombosis will usually not occur if the value for prothrombin is less than 30 per cent of normal. Therein, then, lies the essence of the therapeutic exhibition of dicumarol. The drug must be administered in doses sufficient to hold the patient's prothrombin value within the limits of the individual tolerance, somewhere between 10 and 30 per cent of normal.

After considerable study we have finally adopted the following plan of administering dicumarol. When the drug is definitely indicated and when used prophylactically, 300 mg. are given on the third postpartum day as the first dose. Each subsequent day the prothrombin time of the blood is checked and if the value for prothrombin is found to be less than 20 per cent of normal, no further dicumarol is given, but on the days this value is found to be greater than 20 per cent of normal, an additional 100 mg. are given. If the patient is found to be resistant to the drug, as indicated by only minimal change in the prothrombin time, the subsequent doses may need to be increased to 200 mg. while, conversely, if an unusual sensitivity is demonstrated by the precipitous development of prothrombin deficiency with a prothrombin value that is below 10 per cent of normal, the doses may be further spaced or reduced. Barnes and Ervin[8] reported a study in which they gave 300 mg. of dicumarol while the patient was in labor and 200 mg. on the first postpartum day; they noted no appreciable increase in lochia.

Link[6] showed that the response to dicumarol was slowed and reversed by the simultaneous administration of vitamin K (2-methyl-1,4-napthoquinone. Experience with medical and surgical patients has indicated the prudence of giving 60 to 72 mg. of vitamin K intravenously if the percentage of prothrombin is less than 10 per cent of normal. Previous reports, which concern all types of surgical conditions, show that about 3 per cent of patients receiving the drug experience minor episodes of bleeding, such as petechial hemorrhages of the skin, epistaxis, microhematuria or oozing from raw surfaces, and that about 1 per cent of patients experience severe hemorrhages. It is reasonable to assume that this same percentage of hemorrhagic complications will occur in the postpartum patient if a large enough series is observed, and this risk must be accepted in contemplating the use of the drug.

The contraindications to the use of dicumarol are hepatic diseases with associated prothrombin deficiency, renal insufficiency, states of severe nutritional deficiency, blood dyscrasias with tendency to bleed and subacute bacterial endocarditis. The drug is used only with great caution in patients with ulcerative diseases. Though none of the above-mentioned complications occur frequently in the pregnant or postpartum state, it is well to keep the possibility of their appearance in mind.

CLINICAL EXPERIENCE

Our first experiences with the use of anticoagulants in postpartum patients began in August, 1940, when, within a few days, 2 patients who had been delivered elsewhere were admitted to our service; both had severe pelvic thrombophlebitis. In both of these patients, in spite of the usual conservative treatment, thrombosis developed elsewhere in the body; finally, each had clinical and laboratory evidence of pulmonary infarction and their condition became desperate. It was decided to risk heparinization of these patients, with the realization that should uterine bleeding occur, the situation could probably be controlled with transfusions and withdrawal of the drug. Both patients made an immediate and dramatic recovery. With this experience in mind, heparin and, later, dicumarol when it became available, were then given to additional patients with what we considered satisfactory results.

For the purpose of this study we have reviewed the records of 56 patients with

severe postpartum thrombo-embolic phenomena who have been encountered on the obstetric service at the clinic during the 10-year period, 1937 through 1946. The study by no means included all the cases in which thrombo-embolic phenomena occurred on the service, but in each of these 56 cases the condition was severe enough to assume major importance for the patient. Of this group of 56 patients, 6 received heparin alone, 28 received dicumarol alone, and 2 received both heparin and dicumarol. Thus, a total of 36 patients had anticoagulants of one type or the other or both. Since the remaining 20 patients were treated by means of such conservative measures as warm, wet dressings, elevation of the affected extremity and sedation, it was felt that they might serve reasonably well as a control group.

Studying the records of this group of patients as a whole produced the usual information that is obtained in such cases. About one third of the patients came to delivery with severe anemia. Thirty-three, or 58.9 per cent of the group, had trouble with varicose veins in the prepartum period. Only 23, or 41.1 per cent of the deliveries were spontaneous, while 58.9 per cent were operative deliveries; 17 patients, or 30.4 per cent, were delivered by Cesarean section.

The earliest occurrence of thrombophlebitis was on the third day; the average time of occurrence was the fifth to seventh day. Pulmonary emboli tended to occur about the eighth to the tenth day. In those patients with thrombophlebitis the primary difficulty occurred in the left leg in 30 patients, in the right leg in 14, and in both legs in 6. Nine patients of the entire group had pulmonary emboli. In each instance the diagnosis of pulmonary infarction was confirmed by roentgenogram, and often electrocardiographic evidence was present. Six patients who gave a history of previous pulmonary embolism or severe thrombotic phenomena were given dicumarol prophylactically. We felt that such prophylactic treatment was justified since Barker and co-workers[9] had shown in a statistical study of 678 surgical patients who previously had had nonfatal postoperative pulmonary embolism and infarction that the incidence of subsequent venous thrombosis and embolism was 43.8 per cent, and of subsequent fatal pulmonary embolism, 18.3 per cent. We have reason to suspect that the same approximate percentage relationship may obtain in obstetric patients.

All patients who received anticoagulants made rapid and satisfactory recovery. No patient with thrombophlebitis who received the drugs experienced pulmonary embolism after institution of this type of therapy, though Jorpes[10] reported the expected incidence of pulmonary embolism as a sequel to thrombophlebitis to vary between 15.8 and 35.8 per cent in obstetric cases. The period of hospitalization in the group of patients treated with anticoagulants was less than twenty days for three fourths of the group, averaging about seventeen days for the entire group, and the longest stay was forty-four days.

In the control group only about one half of the patients were out of the hospital in twenty days, the average stay being twenty-two days and the longest stay being sixty-five days. Seventeen of the 20 control patients showed residual venous insufficiency at the end of six months. However, only 5 of 32 patients who were treated with anticoagulants showed any degree of residual venous insufficiency at the end of six months and in only 1 patient was the insufficiency severe. There were no deaths in the entire series of 56 patients.

During the course of our study we encountered an article written by Field[11] in 1945 in which he stated that the rat pups of suckling mother rats which had been given toxic doses of dicumarol succumbed to internal hemorrhages, indicating that the drug was transmitted through breast milk. This gave us some momentary concern for the infants of mothers on therapy, but further investigation showed that surprisingly few mothers in the series had lactated sufficiently to be able to nurse their babies. Studies of prothrombin time

of the blood of the few babies that did nurse indicated that the value for prothrombin was consistently 100 per cent of normal, while the prothrombin time of the mother's blood indicated that the value for prothrombin was between 10 and 30 per cent of normal. We recognize that this may possibly be due to acquired protection, since all of our infants routinely receive injections of vitamin K at birth. However, it would appear that until more is known in this regard it would be safest to discontinue breast feeding for infants of mothers receiving dicumarol, or to give the infants 2.4 mg. of vitamin K intramuscularly every second day and to check the prothrombin time carefully.

In the entire series, 4 patients had minor phenomena of bleeding, usually noted as slight increase in lochia or petechiae of the skin. In no instance was it necessary to do other than to adjust the dose of the drug more carefully. One case which will be reported in detail elsewhere was that of a patient with serious hemorrhage which continued, in spite of two intravenous injections of 72 mg. of vitamin K, until the uterus was packed with iodoform gauze. During the period of bleeding the patient received 7,800 c.c. of blood by transfusion.

In the light of our experience with this patient we are now inclined to believe that should serious uterine bleeding occur during the administration of dicumarol, in addition to giving vitamin K intravenously and sufficient blood to replace the volume of blood lost, there should be no delay in packing the uterus firmly with gauze and in giving oxytocics. These measures, by stimulating firm muscular contractions, tend to close the uterine sinuses and control bleeding in much the same manner that uterine contractions control blood loss immediately after the expulsion of the placenta.

SUMMARY

We have reported on a series of 56 cases in which thrombo-embolic phenomena occurred in the postpartum period. Of this group 20 patients were treated by conservative measures alone and served as a control group. The remainder had anticoagulants of one type or another.

The patients who were treated with anticoagulants made a more rapid, smoother recovery than did those in the control group and they showed almost no residual venous insufficiency when examined six months later. There were no deaths in the 9 cases of pulmonary embolism. The dangers and necessity for close supervision of patients receiving anticoagulants are pointed out.

REFERENCES

1. Davis, A. and Porter, M.: Dicoumarin in treatment of puerperal thrombosis, Brit. M. J., 1:718-719, 1944.
2. Howell, W. H.: The coagulation of blood, Harvey Lect., (1916-1917) s. 12:272-323, 1918.
3. Murray, G. D. W. and Best, C. H.: The use of heparin in thrombosis, Ann. Surg., 108:163-173, 1938.
4. Murray, Gordon: Heparin in thrombosis and blood vessel surgery, Surg., Gynec. & Obst., 72:340, 1941.
5. Roderick, L. M.: Quoted by Link, K. P.[6]
6. Link, K. P.: Anticoagulant from spoiled sweet clover hay, Harvey Lect., (1943-1944) 39:162, 1944.
7. Allen, E. V., Hines, E. A., Jr., Kvale, W. F. and Barker, N. W.: The use of dicumarol as an anticoagulant: experience in 2,307 cases, Ann. Int. Med., 27:371, 1947.
8. Barnes, A. C. and Ervin, H. K.: Effect of anticoagulants on postpartum bleeding, Surg., Gynec. & Obst., 83:528, 1946.
9. Barker, N. W., Cromer, H. E., Hurn, Margaret and Waugh, J. M: The use of dicumarol in the prevention of postoperative thrombosis and embolism with special reference to dosage and safe administration, Coll. Papers, Mayo Clin. & Mayo Foundation, 36:398, 1944.
10. Jorpes, J. E.: Anticoagulant therapy in thrombosis, Edinburgh M. J., 53:222, 1946.
11. Field, J. B.: Hypoprothrombinemia induced in suckling rats by feeding 3,3' methylenebis (4-hydroxycoumarin) and acetylsalicylis acid to their mothers, Am. J. Physiol. 142:238, 1945.

HISTOPLASMOSIS*

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For many years it has been known that throughout a large territory in the Mississippi Valley, pulmonary calcification is a very common occurrence. The generally accepted cause for such lesions has been the tubercle bacillus and the diagnosis "primary complex arrested" is widely used. However, tuberculin testing and x-ray

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studies, too numerous to mention, have produced these conclusions:

1. Calcifications are seen in large numbers of relatively young children who are non-reactors to tuberculin and who are life long residents of low tuberculosis mortality areas.

2. The tuberculin test is very reliable. Sensitivity to the dose .0001 P.P.D. usually persists for years.

These conclusions cannot be reconciled with the view that tuberculous infection is always the antecedent of pulmonary calcification. It is highly improbable that young children can lose tuberculin sensitivity in so short a time. The dose of P.P.D. mentioned above is five times the usual first strength P.P.D. and has been well established as a good critical level for clinical use.[1]

C. E. Smith[2] of Leland Stanford, Jr. University, did a great deal of skin testing with coccidiodin at some of the California air fields because of some slight apparently cross reactions. He came to the conclusion that there was some fungus producing lung infection in the middle west, and he further called attention to the fact that the Mississippi Valley was the endemic area for *Histoplasma capsulatum*. Amos Christie,[3] a former student of Smith's, went to Vanderbilt University in the fall of 1943. In January, 1944, he was able to diagnose histoplasmosis ante-mortem, a very rare procedure at that time. Soon after this he began doing skin tests using a solution of a broth filtrate culture.

Carroll Palmer[4] had been studying minimal tuberculous lesions in nurses in widely separated geographical areas. Routine tuberculin testing and x-rays every six months for a period of over two years had shown wide differences in calcification and sensitivity to tuberculin. This was especially striking when the two cities, Minneapolis, Minnesota, and Kansas City, Missouri, were considered. In each about 1,000 nurses were studied. He found about ten times more pulmonary calcification in the Kansas City group, yet the tuberculosis incidence was about the same in the two cities. Skin testing[3] over 10,000 nurses with 1.1000 histoplasmin solution and tuberculin P.P.D. .0001, Palmer discovered that the two tests picked out practically all of those who had positive findings of pulmonary calcification.

The disease *Histoplasmosis*, has been known since 1905, but it was considered to be a very rare disease. Parsons and Zarofonetis[5] in 1945 published an excellent review of the literature to date and reported 71 cases including their own. The disease at that time was considered to be always fatal. It occurred at all ages but more often in children. In the younger age groups it is characterized by unexplained fever, spleno- and hepatomegaly and leucopenia. It was usually misdiagnosed and called aleukemic leukemia, or some other blood dyscrasia. In the generalized acute forms lung findings are not prominent. In older age groups, lung findings seem more common, but no fixed pattern can yet be given. The lesions found cannot be distinguished from tuberculous by x-ray.

As a result of their studies both Palmer[4] and Christie and Peterson[3] concluded that there must be a very wide subclinical infection with histoplasma and that relatively few cases developed fatal forms. In doing this they were following the course of events that occurred in the study of coccidiomycosis and also for tuberculosis, both of which diseases were at one time considered always fatal. Palmer,[6] continuing his nurse study, showed the geographic distribution of the persons listed. Over 10,000 nurses in eleven different cities were tested. He showed that the areas of greatest incidence ranged from eastern Kansas, eastward, including Ohio, and southward including Arkansas, Tennessee, and northeast Louisiana.

Since the original articles were published a great deal of work has been done, mainly by two groups, Christie and Peterson at Vanderbilt and by Palmer and his co-workers in the United States Public Health Service. In the summer of 1946 a field laboratory of U.S.P.H.S. began operation in Kansas City, Missouri, under the direction of

M. L. Furcolow. From this laboratory have come a number of important contributions. Many thousands of children and adults have been tested and x-rayed. From these studies much has been learned. At the start of this work we had these facts. There are a tremendous number of persons with calcified lesions who do not react to tuberculin and who do react to histoplasmin. There is a definite disease, histoplasmosis, which has been regarded as practically always fatal. It had been postulated that there was a subclinical or mild form of histoplasmosis which produced calcification. There were many missing links to be found.

One of the first questions to be settled was the specificity of the skin test. Among others, Emmons and Olsen[7] question this. There certainly is no doubt but that there are some cross reactions between many of the fungi. Christie and Peterson are sure haplosplorangium is not a factor.[8] The greatest controversy seems to be concerning blastomycosis and histoplasmosis.

Howell[9] studied these in animals and reported that "if the critical titre of these antigens are determined and if these concentrations are used to study cross reactions, the degree of cross reaction between these antigens is small and the antigens are, therefore, relatively specific for guinea pigs experimentally infected with the homologous fungi". In other words if correct doses are used there is little trouble. Furthermore in the studies in Kansas City the failure to find blastomycosis clinically leads us to believe it is not a factor in that area; also it is reported to us that blastomycosis does not produce calcific lesions.[10] Work has not yet reached the point where we can say that the histoplasmin test is as reliable as the tuberculin test, but a part of the way has been traversed.

Another real point at issue was whether or not all proved histoplasmosis cases are fatal. Bunnell and Furcolow seem to have settled this question.[11] They reported 10 proved cases and since their report was written have added 2 more. Five of these have recovered definitely and two others are alive several months after diagnosis, but the prognosis of these two is doubtful. The 5 give every indication of being perfectly well and of remaining so. This gap seems to be definitely bridged.

Another difficulty encountered was the problem of early diagnosis. Routine skin tests may be positive with or without pulmonary x-ray findings and the chest lesions when found vary considerably. Apparently the skin test behaves similarly to the tuberculin test in that during the first few weeks following infection it is not positive and in the presence of overwhelming disease there is no reaction.

Numerous men have been working on a complement fixation test. These reports have been published to date: Salvin[12], Miller *et al*,[13] Tenenberg and Howell,[14] and Furcolow, Bunnell and Tenenberg[15]. The latter two reports deal with the work in Kansas City. It is too early to draw definite conclusions about the value of this test. However, all but one of the known proved cases have been positive. Some of the suspected cases have been positive and over 200 controls have been negative. The test seems to have great value in that it will be positive in those with active disease.

Other laboratory aids have been thoroughly studied, especially blood cultures, sternal punctures, biopsy methods, and others. In several cases the fungus was grown by culturing gastric washings. In patients with generalized disease, blood cultures have been quite successful. In the small or limited lesions and especially those in which some calcium has already been deposited no method of isolating or finding the organism has been successful.

Early in the investigation it was thought that if the pre-calcific lesions were of sufficient severity to necrotize lung tissue they should be visible on the x-ray film. Furthermore if the end result was indistinguishable from a tuberculous lesion, so might the *early* histoplasma infection. To study this over 16,000 Kansas City school children had tuberculin and histoplasmin tests and chest x-rays. All tuberculin reactors were excluded—all types of pulmonary lesions that appeared to be non-

calcified were grouped and restudied. Skin tests were repeated and chest films made at intervals of three or more months. In order to exclude acute infections, all lesions had to persist for two months or over. In addition all cases were given as complete a work-up as possible for tuberculosis, sarcoid, and other infections. From this large group 72 cases were finally selected. These were reported by Furcolow, *et al.*[16] These range from cases with definite lymph node enlargements to those with single and multiple fairly discrete lesions with and without nodes, to massive and multiple infiltrations occupying both lung fields. Some of these cases have been followed over two years and a characteristic calcifying process observed.

Tuberculous lesions have been observed and their course described by many. Beginning with tuberculin non-reactors, the reaction has been seen to change, characteristic pulmonary lesions appear, and then gradually shrink and calcify. Various parts of this cycle have been seen to occur in patients sensitive to histoplasmin but who are tuberculin non-reactors. In the nurse studies, some non-reactors have changed to reactors and infiltrates in the lungs have been observed but not enough time has elapsed to see the final picture. In 2 of the cases in the group of 72, multiple diffuse lesions were present in both lung fields. From each of these cases histoplasma was isolated. Each has now progressed through the stages of clearing, resolution and calcification so that the end seems not far off. Both will have the final picture commonly described as "wheatena," with hundreds of small calcified areas in each lung. Most of the cases found will not have such extensive disease. There will be only one or two small areas of soft infiltration. In such cases it is doubtful if the disease ever extends further than the adjacent nodes and the organism does not circulate in the blood stream. Since these cases are not clinically ill and the disease seems to have no effect whatever it has been extremely difficult to obtain any pathologic material. Surface lymph nodes seem to be rarely involved. Therefore, there are gaps in our knowledge which have to be filled.

However, if there are gaps yet existing, enough evidence is at hand to be of definite clinical assistance. First of all, we are now more sure of the reliability of the tuberculin test if employed in low dosage. We feel sure that these children with soft infiltrates and calcified or partially calcified areas, who are not tuberculin reactors, do not have tuberculosis. So far we have seen no reason for altering the daily routine of these patients. This is very important because in the past many of them were put to bed for long periods of time and many children and parents were unduly alarmed. In older age groups, for example, in the late teens soft infiltrates are seen which cannot be distinguished from tuberculous lesions by x-ray. It is the practice of some men to do collapse therapy on such minimal lesions. In the areas of histoplasmin sensitivity, these lesions must be carefully considered, and collapse therapy not done unless the tuberculin test is positive. In my opinion pneumothorax should not be done unless tubercle bacilli are found.

In adults, the picture is more confusing. Hollis Johnson[17] of Nashville has reported a patient with a definite cavity. *Histoplasma capsulatum* was found repeatedly in the sputum. The lesion had all the appearance of a tuberculous lesion but tubercle bacilli were never found. A similar case is now under observation in Kansas City. Another case with chest lesions typical of tuberculosis came to autopsy. Diagnosis of histoplasmosis had been made by culture and tissue examination following splenectomy. Blood cultures and gastric cultures were positive. At autopsy, histoplasma was demonstrated in many organs and also tubercle bacilli were found in the lungs. In this case it was difficult to say which caused the lung lesion. The diagnosis of chest disease has in this area become a question of definitely finding the etiologic agent. There are undoubtedly many

cases of histoplasmosis masquerading as tuberculosis.

There is much more to be learned about the disease. It is significent that as late as January, 1945, a most thorough report collected about 70 cases of the disease. Furcolow and Bunnell in a little over two years time have 12 proved cases. Much has been accomplished to clarify pulmonary diagnosis and it is most likely that in the near future the remaining gaps in our knowledge will be filled.

CONCLUSION

A brief summary of the principal facts and theories of histoplasmosis as they exist at this time are given. The histoplasmin skin test if properly given, seems to be a reliable test. The complement fixation test has great promise. Known cases of histoplasmosis are alive and well. The necessity of careful differential diagnosis for tuberculosis is emphasized as some proved cases of histoplasmosis have all the appearance of tuberculosis. The possibility of coexistence of the two diseases must be born in mind and to date there is no information in regard to symbiotic phenomena. Northeast Louisiana is an area of high incidence for histoplasmin sensitivity.

REFERENCES

1. Furcolow, M. L., et al: Quantitative studies of the tuberculin reaction; I. Titration of tuberculin sensitivity and its relation to tuberculous infection, Public Health Reports, 56:1082-1100, 1941.

2. Smith, Chas. Edward: Coccidioidomycosis, Med. Clin. North America, 27:900, 1943.

3. Christie, Amos, Peterson, J. C.: Pulmonary calcification in negative reactors to tuberculin, Amer. J. Pub. Health, November, 1945.

4. Palmer, Carroll E.: Non-tuberculous pulmonary calcification and sensitivity to histoplasmin, Public Health Reports, 60:513, 1945.

5. Parson, Robert J. and Zarafonetis, C. J. D.: Histoplasmosis in man, Arch. Int. Med., 75:1, 1945.

6. Palmer, Carroll E.: Geographic differences in sensitivity to histoplasmin among student nurses, Pub. Health Reports, 61:14, 1946.

7. Emmons, C. W., et al: Studies of the role of fungi in pulmonary disease; I. Cross reactions of histoplasmin, Pub. Health Reports, 60:47, 1945.

8. Christie, Amos and Peterson, J. C.: Pulmonary calcification and sensitivity to histoplasmin, tuberculin and haplosplorangin, J. A. M. A., 131:658-660, 1946.

9. Howell, Arden: Studies of fungus antigens, Pub. Health Reports, 62:13, 1947.

10. Smith, David T., Duke University: Personal Communication.

11. Bunnell, Ivan L. and Furcolow, M. L.: A report of ten proved cases of histoplasmosis, Pub. Health Reports, 63:10, 1948.

12. Salvin, S. B.: Complement fixation studies in experimental histoplasmosis, Proc. Soc. Exp. Biol. and Med., 66:342, 1947.

13. Miller, H. E., et al: Histoplasmosis, etc., Arch. Derm. and Syph., December, 1947.

14. Tenenberg, Daniel J. and Howell, Arden: A complement fixation test for histoplasmosis, Pub. Health Reports, 63:6, 1948.

15. Furcolow, M. L., Bunnell, Ivan L.: Tenenberg, Daniel J.: A complement fixation test for histoplasmosis, Pub. Health Reports, 63:6, 1948.

16. Furcolow, M. L., Mantz, Herbert L., and Lewis, Ira: The roentgenographic appearance of persistent pulmonary infiltrates associated with sensitivity to histoplasmin, Pub. Health Reports, 62:49, 1947.

17. Johnson, Hollis: Unpublished Report. Paper read at A. C. C. P.—Southern Chapter, Baltimore, November, 1947.

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PEDIATRIC SURGERY*

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NEW ORLEANS

Compared with the tremendous volume of surgery done for adults, operations in infancy and childhood are not common. In general they are rare. Even from the children's hospitals, leaders in surgical advances in this field report tens and hundreds of operations of a specific type upon children whereas thyroidectomies, colon resections, hysterectomies, and other formidable procedures are reported even by individual surgeons in terms of thousands. The infrequency of surgery in infancy has resulted in a lack of experience in this field both in surgical technic and in surgical diagnosis. Most of the surgical problems of childhood will have to be handled by the general surgeon. How many general surgeons, extremely able and well informed otherwise, know immediately the answers to many of the questions pertaining specifically to the surgery of infancy; for exple, the total parenteral twenty-four hour fluid and salt requirement for a baby with congenital atresia of the intestine or a pyloric stenosis? The answer to a similar question regarding adults is standard information known to every surgeon.

Technical and diagnostic advances have been made and the scope of surgery of in-

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fancy is rapidly being expanded. The type of surgery requirement encountered in infants differs sometimes from that of adults. Many other malformations may await correction at a later time but the serious obstructive malformations of the gastrointestinal tract require immediate operation. Alleviation or correction of obstructive lesions, such as hypertrophic pyloric stenosis, the operation for which is the most common operation performed in infancy, is imperative surgery. Imperforate anus and atresia of the esophagus likewise demand immediate surgical intervention. Tracheoesophageal fistulas are stated to be almost as common as hare lip, but this defect is frequently overlooked and it may cause death from pneumonia with the fundamental cause being unsuspected.

Since hypertrophic pyloric stenosis is the most common lesion requiring major surgery, I wish to recall a few important features in its management. Hypertrophic pyloric stenosis usually begins to manifest itself at from the third to the sixth week of life. It is characterized clinically by eructation and vomiting increasing in severity and by weight loss. Objectively an olive-sized mass is usually palpable in the epigastrium. Roentgenography shows gastric dilatation and retention and a funnel-shaped obstruction at the pyloric, with a string sign, an attenuated trickle of barium seen in lateral roentgenograms distal to the funnel-shaped obstruction.

A Fredet-Rammstedt operation is usually indicated when the diagnosis is established. The surgical risk is less than 1 per cent, whereas medical management alone is attended by a mortality of from 10 per cent to 30 per cent depending on the number of cases withheld from surgery.

These infants are dehydrated and depleted of proteins, and they are prone to develop a complication which is far too common following this operation; that is, evisceration. There is a great tendency to evisceration in infants. Even experienced surgeons may encounter it in 1½ per cent of cases. (Ladd and Gross). The inexperienced surgeon may feel that since it is a child, wound healing will be rapid, and therefore, he will remove the stitches early. The result may be an evisceration. To prevent evisceration certain precautions are important: Fluid and proteins should be replaced as far as possible to the normal level; a transverse incision should be made, or if a right rectus incision is made it should be placed high so that during the period of healing the liver will protect the wound. I prefer retention sutures and do not like the sutures removed early (not under ten days). Another important technical feature of the operation is to sever the pyloric muscular ring and permit the mucosa to bulge without opening into the lumen. Such a misstep is more likely to occur at the duodenal end where the hypertrophic pyloric sphincter ends abruptly. The surgeon should be extremely cautious in that area.

Among the other types of operations required in infancy are those for atresia or stenosis of the duodenum or jejunum. Such an obstruction manifests itself promptly at the beginning of life. The predominant symptom is vomiting. The vomitus contains bile. Roentgenograms show air in the stomach and duodenum and no evidence of gas in the lower bowel. A roentgenogram after instilling lipiodol or perhaps barium will usually show a dilated stomach and duodenum. (Fig. 1) Congenital obstruction of the duodenum notably occurs in the distal portion. Stenosis simulating complete atresia frequently is due to compression of the duodenum by peritoneal bands. A true atresia may be difficult to differentiate clinically from a stenosis due to such a peritoneal fold or to obstruction from malrotation of the intestine. Peritoneal veils causing obstruction may be found either across the third or fourth portions of the duodenum or across the first portion of the jejunum and should be searched for very carefully when duodenal stenosis or atresia is established as the cause of the vomiting. If there is a peritoneal veil, it may be cut and released and thus the obstruction is obviated. On the other hand, if there is a true atresia of the duodenum a gastro-en-

terostomy is usually conceded by most authorities to be the operation of choice. The roentgenogram in *Figure 1* shows a dilated

Fig. 1

Roentgenogram of a one month old baby showing obstruction at the end of the duodenum. The child presented signs of high partial intestinal obstruction. At operation a veil of peritoneum compressing the first part of the jejunum was found. This was severed and the child recovered promptly, showing no further symptoms of obstruction.

duodenum and stomach. Vomiting began early in life and conservative management resulted in weight loss and emaciation. At operation, performed when the child was one month old, peritoneal bands were found obstructing the jejunum at its initial segment. The obstructing bands were severed and the child promptly recovered.

Other obstructions of the gastrointestinal tract involve those near the anus or rectum. The Wangensteen-Rice method of determining the distance of the rectal pouch from the anal dimple should be well known and constantly used. When the rectal pouch is very high a colostomy is indicated. Fortunately this type is not common. Various fistulas into the urinary tract or in the female between the rectum and the vagina are associated with atresia of the rectum (55 per cent of cases). These should be sought for carefully and recognized. Usually establishing a lumen through the normal area of the anal canal is a procedure of choice and the fistulas if present may secondarily close spontaneously or be closed by further operative procedure.

Atresia of the esophagus with tracheo-esophagus fistulas are by no means as rare as they were formerly thought to be. These are very complicated surgical problems. Many of these patients can be saved and returned to normal life by prompt operation. Many undoubtedly die of pneumonia without the true foundation of the cause of death being known. Establishing the diagnosis of the tracheo-esophageal fistula depends upon roentgenographic findings. In the gastrointestinal tract, there will be no air shown in those instances in which the lower segment of the esophagus does not communicate with the trachea. If there is a tracheo-esophageal fistula between the upper esophageal segment and the trachea (a rare type), lipiodol instilled into the esophagus will make its appearance in the lung field. In the more common type there is a communication between the lower segment of the esophagus and the trachea, and in roentgenograms of the abdomen air is found in the intestine. Lipiodol instilled into the esophagus shows a blind pouch and possibly some lipiodol aspirated into the trachea. Lipiodol does not make its appearance in the stomach. A number of these children have been saved by direct end-to-end anastomosis. Occasionally there is too much separation of the upper and lower segment of the esophagus. A palliative procedure disconnecting the fistula and a gastrostomy with a later operation to reconstruct an esophagus is a method which has been used for salvaging other children.

Various orthopedic malformations may present themselves early in life. Often these require corrective operations or corrective management over a long period of

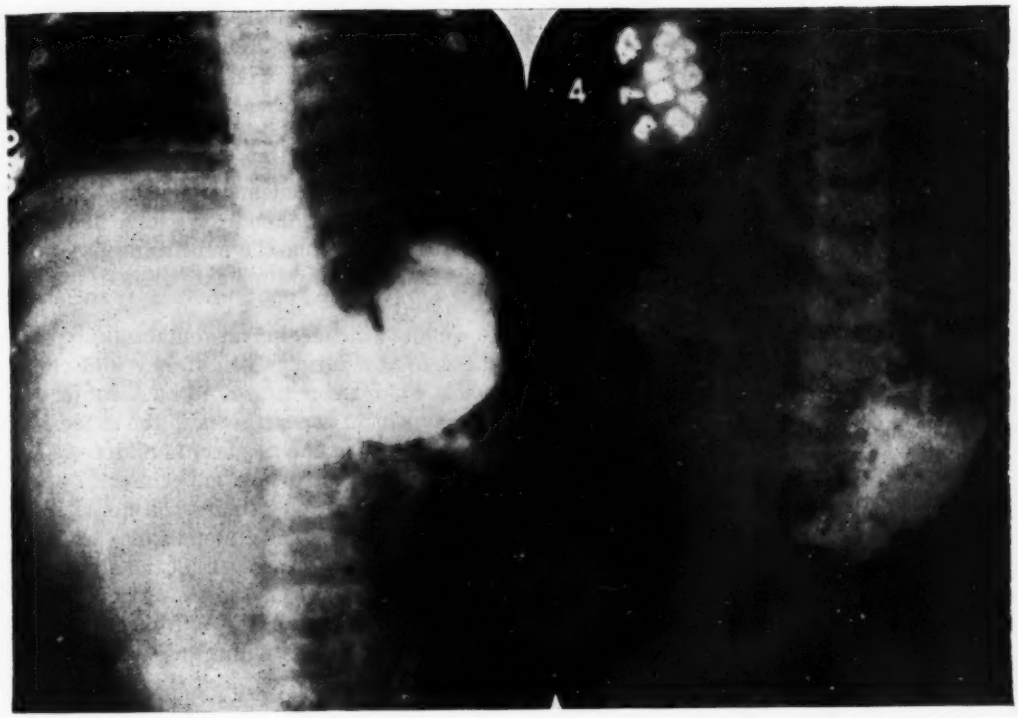

Fig. 2

Marked displacement of the gastrointestinal tract to the left side of an abdomen in a 16 month old child. The tumor causing this was connected with the right kidney. Nephrectomy was performed and the child is still living and apparently well one year following operation. About 25 per cent of children with embryomas of the kidney (Wilms' tumor) may be salvaged by surgery. Other lesions which may thus displace the gastrointestinal tract are omental and peritoneal cysts, enterongeous cysts, tumors of the liver and idiopathic dilatation of the common bile duct.

time. Usually an operation is not imperative. Urologic malformation such as extrophy of the bladder are also distressing developmental defects which may wait beyond the initial period of infancy before the correction is attempted.

Many embryologic tumors are found in infancy among which should be mentioned the embryomas (Wilms' tumor) of the kidney, and various teratomas. These require prompt removal. A Wilms' tumor usually presents itself as a mass on one side of the abdomen. (Fig. 2.) Gastrointestinal tract roentgenogram shows that the gastrointestinal tract has been displaced remarkably to the other side. Pyelograms may show distortion indicating that the tumor involves the kidney. Usually Wilms' tumors are highly malignant. On the other hand, about 25 per cent of the children might be saved by a nephrectomy. The proper nephrectomy involves a transabdominal approach with primary ligation of the vein and arteries to the kidney. The mass may then be removed without the same fear of disseminating the malignancy through the vascular system while the tumor is being manipulated. Other infantile lesions which displace the gastrointestinal tract are omental and mesenteric cysts and congenital dilatation of the common bile duct. Various other cysts and tumors involving the abdominal cavity, and masses caused by duplications of various segments of the gastrointestinal tract, may be encountered in infancy. Fortunately they are

rare, but they may require removal early in life.

Among the congenital defects in infants and children are hernias. Inguinal hernias, of course, are very common in the male. They may be repaired early but the optimum time is probably after the second year of life. Occasionally a strangulation or large hernia demands a repair during infancy. Usually there is nothing so difficult about repairing these congenital inguinal hernias but the tissues are extremely delicate. Omphalocele, large umbilical hernias, and congenital diaphragmatic hernias, if present, demand prompt recognition and emergency operation. A useful bit of knowledge in the operation for omphalocele is, that if the protrusion of abdominal viscera is marked and closure of muscle layers over the viscera can be accomplished only with undue tension, closure of the skin alone over the viscera may be a life saving measure. A two-stage repair of the abdominal wall is accomplished, the muscle layer being approximated aften ten to fourteen days.

Diaphragmatic hernias, if present, may cause extreme dyspnea. Roentgenograms may disclose the cause for this. Usually the diaphragmatic hernia develops through the pleuroperitoneal canal, the foramen of Bochdalek. This is a posterior lateral aspect of the diaphragm where the anterior and posterior muscular layers normally fuse. As in the adults the preliminary phrenic nerve crushing, with immediate abdominal approach after decompressing the stomach and gastrointestinal tract usually permits a satisfactory closure. Ladd and Gross state that these hernias may be repaired much more easily in infants in whom the gastrointestinal tract had not had time to expand from interluminary air and ingested contents. If the lesion is diagnosed early in the infancy, it should be repaired immediately.

Various vascular abnormalities including patent ductus arteriosus and the tetralogy of Fallot have been yielding to recent marginal advance in the surgery of infancy and childhood. The outstanding work of Gross and Blalock in this respect is becoming widely known not only to the profession but to the laity as well. These lesions usually do not demand emergency surgery, but they may require surgery during childhood after the period of infancy has passed. Congenital lymphedema should be operated upon early in childhood. We have had good results by excising the subcutaneous tissue and fascia containing the dilated lymphatics and fat.

Among the technical outstanding features in the surgery of infancy and childhood, what never ceases to be a subject of amazement to the surgeon, is the extremely fine delicacy of the tissues of children. Anyone who has performed experimental operations on dogs and rabbits has noted their transparently delicate membranes and organs and one is impressed by the similarity of the consistency of the tissues of the infant and of the rabbit. Both of them have very exquisitely fine organs. The small gastrointestinal tract and thin walls require very careful handling. Because of this, it is essential for a surgeon to be careful in making incisions in an infant. It is very easy to overcut particularly when customary force and speed has been used on the morning busy with adult surgery. In doing hernioplasties for example in small infants the external oblique aponeurosis appears so thin and frail that it would not seem capable of retaining the pressure imposed upon the abdominal wall. Carefully placed fine sutures are essential in this type of surgery. In making anastomoses or correcting pyloric stenosis, gross estimates of incisions and movements are not enough; they must be calculated almost with micrometer accuracy to be correct. For that reason it behooves the surgeon handling operations of infancy and childhood to be more cautious than he would be in his own experienced field of adult surgery.

A word about fluid requirement of infants. Almost all surgeons know how much fluids their adult patients require every day. This may be calculated even for difficult problems such as obstructions at the pylorus or upper gastrointestinal tract. The effort of replacing fluid and salt in the

quantity to be used is relatively well known in adults. On the other hand, the fluid balance and fluid requirements of infants is a more difficult subject. Usually an infant requires for period of twenty-four hours from 70 to 90 cubic centimeters of fluid for each pound of weight. A maximum sodium chloride requirement is 4 or 5 grams. In an adult thus an intake of 2500 to 3000 cc. of fluid a day is comparable to an infant's intake of from 500 to 600 cubic centimeters a day. In addition to that, the infant will not be able to use sodium chloride to the same extent as an adult. A normal requirement for an adult would be less than 10 grams a day, but for an infant 4 or 5 grams is abundant and an intake of 10 grams of salt a day will eventually result in salt edema. Transfusions in infants may be given in the head or neck veins and a quantity of blood equivalent to 10 cc. for each pound of body weight would be comparable to a 500 to 700 cubic centimeter transfusion in an adult. Sometimes in dehydrated states after repeated vomiting an intake in an infant of as much as 1000 cc. a day may be correct. Such a daily intake must not be continued otherwise the child will develop edema from plethora. Children should be prepared for operation carefully just as in adult life. Such preparation necessitates regard for the same factors of fluid and electrolyte replacement, protein and blood replenishment.

Only by careful attention to all the details will good results be attained for these children. After all, if there is any great responsibility about doing surgery it probably increases inversely with the age of the patient. The loss of an old man or an old woman is not nearly so regretful as the loss of a baby or a young child.

UNUSUAL MANIFESTATIONS OF CORONARY HEART DISEASE*

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The recognition of coronary heart disease in all of its clinical manifestations is the ultimate aim of all clinicians. We are all familiar with the typical cases described in our textbooks, such as those with substernal pain radiating to the left shoulder and arm; those suffering with angina pectoris which is relieved by nitroglycerin; the individual with a severe substernal or precardial pain with its associated drop in blood pressure, elevated white count and a slight elevated temperature as in coronary thrombosis; and also, the acute pulmonary edema of the congestive heart failure. These cases, which are so well described in our textbooks, and with which all of us are very familiar, unfortunately do not cover all of the cases that we encounter in our practice. Some of the manifestations, less common than the classical ones, are those in which we are interested at this time.

An effort to get to the root of an obscure symptom, particularly symptoms arising from the heart, requires a very detailed history, and by process of elimination symptoms are ruled out one at a time, after which we resort to our physical examination and usual laboratory aids. Details are obtained, such as symptoms being brought out following effort, during exposure to cold weather, etc. Because of the many possibilities in the differential diagnosis, a short cut can sometimes be obtained from the family history, which might immediately unveil the picture of a possible cardiac condition because of a well known tendency for certain families to develop cardiovascular disease.

In addition to the history, the physical examination will frequently reveal some clues that might clear up a questionable situation in the absence of definite blood pressure changes and obvious heart abnormalities. The middle-aged, square-chested individual is well known to all of us as well as the obese patient. One observation from the physical standpoint which might bear more detailed study is the prematurely grey individual, of whom I had two very recently who turned out to have myocardial disease.

One of the most difficult situations to

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analyze is that in which the patient complains only of weakness. This symptom can be extreme and the patient will frequently state that the slightest exertion makes him feel as though he had done a hard day's work. It is a symptom similar to that which we encounter in a post-pneumonic state or immediately following moderately severe or severe influenza. During the course of any illness a complaint of weakness beyond the usual expectancy in that case should lead one to suspect the possibilities of heart disease. A fainting spell in a previously well individual frequently may be the first sign of heart trouble.

Pain is by far the most common complaint at the onset of coronary thrombosis. The majority of these patients will leave no doubt in your mind when the location of pain is substernal, precordial, or across the epigastrium. Apparently pain can radiate to almost any part of our anatomy. Some individuals with coronary heart disease may manifest gastrointestinal symptoms only, complaining of epigastric pain, pain in either upper quadrant of the abdomen even to the extent of having a rigid upper abdomen to make you think of a ruptured ulcer, as in one case that I remember so vividly. A delay for observation and the development of a friction rub saved this man an exploration. Gallbladder disease is frequently found in a cardiac patient. Pain from the gallbladder will often be radiated substernally. Shoulder pains frequently offer a challenge to your diagnostic ingenuity, particularly these individuals with an acute bursitis or calcified tendons which causes a pain not only in that site but involving sometimes the whole upper half of the chest. Recently, I had occasion to see a man with bilateral calcified subdeltoid bursitis and calcified tendons with severe pains across the upper chest. The idea that all chest pains are cardiac in origin until proven otherwise is a good idea, provided adequate steps are taken to prove or disprove the possibility of heart disease. It might be best to say that all chest pains are not due to heart disease, certainly for the benefit of the patient, as he is already convinced that he has something wrong with his heart. Because of the obscure radiation of pain it is safer to consider chest pains as cardiac in origin unless you can readily account for such pain at the time of your initial examination. The radiation of pain to the lower extremities is not uncommon and should be considered even in such conditions as sciatica, as we have known of sudden deaths during the course of observations or treatment for such pains, which at autopsy revealed the presence of a coronary thrombosis which had apparently existed for some period of time.

I would like to present briefly some cases which were obscure at the onset and after observation and detailed study revealed the true nature of the illness.

CASE REPORTS

Case No. 1. A white female, age 55, consulted a physician because she had had an attack of indigestion the night before and was still a bit uncomfortable. The indigestion consisted of a feeling that she had a "knot in the pit of her stomach," and a great deal of belching. She left the physician's office with a prescription for one of the more popular anti-spasmodics and within a few days the symptoms disappeared. Two months later she was seen by me for a routine health check and an electrocardiogram made at that time showed pathognomonic evidence of an anterior myocardial infarction. No treatment was offered except advice as to limitation of activities.

About six months later this same patient suddenly felt very faint and suffered an attack of syncope accompanied by a profuse cold sweat. She was seen in this state within fifteen minutes of the onset of the attack. Her pulse was of low tension and poor volume and the blood pressure was 90/60. She had no pain and was under the impression that she had simply fainted. An electrocardiogram was made immediately. It showed pathognomonic evidence of a fresh posterior myocardial infarction. The subsequent course was typical with fever and leukocytosis.

This patient then had two attacks of myocardial infarction, one without any pain at all, the other with gastrointestinal symptoms of such a nature that neither the patient nor the physician considered that they might be cardiac.

Case No. 2. A white male, age 58, was admitted to the hospital complaining of paroxysms of cough and marked weakness. For a period of four months he had had a severe sinus infection and was under treatment. A mild cough which had accompanied the sinusitis became more severe one week before he came under observation. When first

seen this patient appeared disorientated, temperature was 101° F. blood pressure 88/50, pulse rapid and of poor volume. Coarse rales were heard throughout the whole chest. A blood count revealed 20,000 white cells. An electrocardiogram revealed evidence of posterior coronary infarction. On the second day of hospitalization he developed an auricular fibrillation which cleared up after forty-eight hours. Ten weeks later he had recovered sufficiently to be allowed out of bed. This patient has never complained of pain. His only complaint throughout his illness was of weakness.

Case No. 3. A white male, 27 years of age, reported to the office with a complaint of pain in the chest on exertion. The pain lasted five to ten minutes and was relieved by rest. The symptoms were so typical that a layman advised him to consult a physician because "it sounded to him like he had angina". Closer questioning brought out the following past history: While the patient was in basic training in the Army he became ill suddenly one day with pain in both shoulders. By next morning he had temperature of 102.5° F. and was put in the station hospital. The pain in the shoulder continued. X-rays were made of his chest as well as all sorts of blood tests. The elevated temperature disappeared in two weeks and he was discharged from the hospital one week later. He was told by the medical officer that they had never found out just what had been wrong with him. He was returned to duty and then served in the European theater as a combat infantryman for over a year, having no difficulty except slight pain in the shoulders during time of great excitement. This pain he paid no attention to at all.

The electrocardiogram made in the office showed pathognomonic evidence of an old posterior myocardial infarction. There can be little question but that the obscure illness which he had during his basic training was a myocardial infarction and that he served as a combat infantryman for over a year after that event.

Case No. 4. A white male, 39 years of age, was first seen in consultation February 20, 1948, at which time he complained of substernal pain on effort. This condition had existed for approximately sixteen days prior to our first seeing him. His physican had prescribed nitroglycerin because of the suspicion of a probable angina pectoris. There were no other complaints. A blood count was normal. Blood pressure was 128/88. An x-ray of the chest revealed normal findings. An electrocardiogram failed to reveal any abnormality. Two days after first seeing the patient he was hospitalized because of severe pain in the left upper chest with point tenderness in the second interspace in the anterior axillary line. This pain was constant, was accentuated by pressure and movement of the left shoulder girdle caused more acute pain. A few days after admission to the hospital he developed acute pain between the left axillary lines and a friction rub was detected. There were symptoms suggestive of a diaphragmatic pleurisy and the clinical course resembled a lower left lobar pneumonia. An x-ray of the chest at this time revealed evidence of consolidation of the lower left lobe. A few days later the patient died very suddenly. An autopsy revealed a marked thinning out of the myocardium at the apex of the heart and a soft scar in this thinned out area indicating a recent coronary thrombosis. Attached to the wall of the left ventricle were many small clots which probably accounted for the pathological report of pulmonary embolism.

A thorough study consisting of a good history and physical examination along with laboratory aids such as blood counts, electrocardiograms, venous pressure, vital capacities, etc. will often clarify a situation which would otherwise be missed. The reliance on diagnostic points is important. The reliance on one or more negative findings, as for instance in using the electrocardiogram, frequently leads to grave errors. In one of the cases mentioned above the diagnosis was made before the autopsy, because of the possible causes of sudden death in such a case.

SUMMARY

Individuals previously in a good state of health, or who during the course of an illness manifest unusual weakness; or patients with obscure chest and upper abdominal complaints, which cannot be readily accounted for, should be carefully studied from the cardiac standpoint.

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THE SURGICAL MANAGEMENT OF UTERINE RETRODISPLACEMENTS

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During the past decade or more surgical treatment of uterine displacement has apparently fallen in popularity. It is the judg-

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ment of these authors that this fallen popularity is unjustified. When the surgical correction is properly applied, it has a very definite place in the treatment of uterine displacement.

With this idea in mind, one of us reviewed the charts of all the uterine suspensions done in Charity Hospital during the past five years, 1943 through 1947. The cases represent all of the three services and were done by many operators, visiting and resident.

This study was undertaken to ascertain the number of operations, the indications for surgery, and the final results as to successes or failures.

There were 219 uterine suspensions done, but 26 were employed incidental to a surgical procedure on the adnexa. These cases are excluded from the study. The remaining 193 cases are reviewed.

In this period there were approximately 6250 major gynecological procedures. Uterine suspension comprised 3 per cent of the operations.

TABLE 1

RACE AND AGE
TOTAL 193 CASES

White—125	Colored—68	
Number	Age	Per cent
10	14-19	5
136	20-29	70
46	30-39	24
1	42	5

Average age—25

Table I lists the cases according to race and age. One hundred twenty-five were white and 69 were colored. The low incidence in the colored race can be explained on the basis that in the negro pelvic inflammatory disease and myomata are frequently seen, and it is unusual to be able to handle such cases with conservative surgery; 136 or 70 per cent of the patients were between the ages of 20 and 29. The limits were 14 and 42, while the average was 25 years which is a bit lower than the average reported by Gardner on his private cases.[1]

The chief complaints of all the patients are shown in Table 2. It was rare to have only one complaint. The three most commonly associated were low abdominal discomfort, backache, with dysmenorrhea and menstrual disturbances following in close order.

TABLE 2

CHIEF COMPLAINTS

Low abdominal discomfort	144
Backache	118
Dysmenorrhea	54
Menstrual disturbances	52
Stress incontinence	26
Dyspareunia	18
Vaginal discharge	16
Sterility	5

There are over a hundred different types of suspensions. The most popular type at Charity Hospital is the modified Gilliam (Table 3) in which the round ligaments are brought through the internal abdominal rings and sutured to the under surface of the anterior rectus fascia. In addition, however, to the procedure on the round ligaments, the uterosacrals are plicated onto the posterior surface of the uterus. This combined procedure was employed in 81 per cent of the cases.

TABLE 3

TYPES OF SUSPENSION

Modified Gilliam with plication	157	81%
Modified Gilliam without plication	31	
Norris suspension with plication	2	
Reefing round ligament	1	
Baldy Webster with plication	1	
Gilliam without plication	1	

In order to evaluate the results of the treatment, the cases were divided into the methods of handling so that we could determine whether suspension alone is sufficient to relieve the suffering patient. The criterion for good results was complete relief from all complaints. Follow-up period averaged eleven months for all patients. Naturally those done in 1947 did not have this average follow-up except those in the first part of the year. The results are informative (Table 4); 74 per cent or 62 patients who had plastic repairs in addition to uterine suspension were relieved; whereas only 66 per cent or 36 patients were cured who had suspension alone. We believe the difference is significant.

TABLE 4
193 CASES RETROVERSION
Results according to handling

	Total-cases		Good		Failure	No follow-up
Suspensions only	54	27%	36	66%	13	5
Suspension with trt. to cervix	38	18%	25	65%	8	5
Suspension with procedure on adnexae	9	4%	5	55%	3	1
Suspension with presacral neurectomy	9	4%	5	55%	3	1
Suspension with plastic	83	43%	62	74%	19	2
TOTAL	193		133	68%	46	14

Next we decided to investigate the reasons for the failures, and these are listed in Table 5. Of the 46 failures, 23 or 50 per cent were failures to correct prolapse and/or relaxations. In other words, the surgery was not adequate. Eight failures were seen in nulliparous patients who had no previous trial with a pessary. In this group of failures was the fourteen year old girl who only had a retrocession. Certainly in this case it is felt that a suspension procedure was not indicated.

TABLE 5
REASONS FOR FAILURE

Failure to correct prolapse and/or relaxations	23
Nulliparas without pessary trial	8
Unknown	6
Cervicitis not treated	5
Pessary gave no relief	2
Symptoms due to orthopedic condition	1
Round ligaments too tight	1

It was interesting to note that only one case of endometriosis was seen at the time of laparotomy in all these cases. This condition is said to be associated often with uterine retroversion; yet it is seldom seen at Charity Hospital.

DISCUSSION

The number of operations, 193, is relatively small (3 per cent) when one considers the total number of other major gynecological operations performed during this same period of time, and apparently the surgery was only used in that small number of cases in which permanent retrodisplacement resulted from structural damages to the fixed and sustaining elements that keep the uterus in its normal position. This surgery is intended to give prompt and permanent relief when pessaries fail to hold the uterus because of inadequate structural supports, or for any other reason that makes the pessary inadvisable.

It is our contention that the disfavor which this operation is enjoying results from the misapplied term round ligament suspension, or plication, leaving with the surgeon the idea that plication or suspension of the ligaments will correct permanent displacement. A better understanding of the factors involved that keep the uterus in a position of anteflexion would at once lead to the discarding of all simpler plication or suspension procedures as totally inadequate.

The uterus is mobile; its normal position is one of moderate anteflexion, but it may change its position, for various reasons, within an arc of 180°. The uterus is suspended and supported in this position by a mechanism which consists of an upper or suspensory level, the round ligaments and broad ligaments. The round ligaments, composed of connective tissue and muscle fibers, extend from the cornual end to the inguinal canal and serve as the ridge-poles of the tentlike structure known as the broad ligament. These two structures, round and broad ligaments, merely suspend but do not support the uterus.[2]

The true supporting structures[3] in the lower one-third of the uterus consist of anterior pubocervical fascia, posterior uterosacral, and the lateral cardinal ligament which fuse with one another and form an elastic sling for maintaining the uterus in its normal position. The uterus is further supported by the perineal body which maintains the normal posterior curve of the vagina, which curve, if not maintained, will allow the uterus to descend carrying its supporting structures along with it.

It therefore can be seen that any opera-

tion merely manipulating the broad or round ligaments in any manner whatsoever —shortening, plication, reduplication—cannot possibly maintain the uterus in the normal position if the more important structures are deficient. The inadequacy of the so-called suspension operations has been responsible for many of the failures and complications that have resulted from their use.

Surgery is only indicated in the acquired symptomatic retroversion, and in all cases the complete operation should include the restoration of the pelvic floor, perineorrhapy; the plication of the pubocervical fascia in order to correct the relaxed anterior and posterior wall; shortening of the cardinal ligaments by plication of the anterior aspects of the uterus or by intra-abdominal plication, as may be most convenient. Shortening of the round ligaments by some sort of suspension is of some help. In many instances the bladder peritoneum may be advanced high on the fundus of the uterus in order to help maintain the anterior position of the uterus.

SUMMARY

1. One hundred ninety-three cases of uterine suspensions at Charity Hospital during 1943-1947 are reviewed as to indications for surgery, and the final results as to successes and failures.

2. A brief discussion as to the supporting and suspending structures of the uterus is given.

CONCLUSION

Prompt and permanent relief of symptomatic acquired retroversions can be attained by surgery if adequate surgery is done. This consists in correcting the retroversion by a suspension procedure and also by correcting the associated damage to the real supporting structures of the uterus, namely, the cardinal and uterosacral ligaments, the anterior pubocervical fascia, and the perineal body. Mere plication or shortening of the round ligaments is not complete surgery.

We wish to acknowledge our thanks to Conrad Collins, M. D., and Adolph Jacobs, M. D., who allowed us to review the charts of the Tulane and Independent services, respectively.

REFERENCES

1. Gardner, George H.: Am. J. Obst. & Gynec., 30:596, 1935.
2. Miller, H. E.: New Orleans M. & S. J., 95:15, 1942.
3. Mengert, William F.: Am. J. Obst. & Gynec., 31:775, 1936.

DISCUSSION

Dr. Howard P. Hewitt (Lafayette): I think the doctor's paper is very timely and I do not like to disagree with the professors, but this is one time that I am compelled to disagree with them. I would like to give you the opinions of a one gallous, two by four country doctor.

What is it that maintains the poise and position of the uterus?

1. The ligaments:
   a) Broad
   b) Round
   c) Uterosacral
2. Intra-abdominal pressure.
3. Paracervical, paravaginal fascia.
4. Pelvic floor.

Since the uterus is only fixed at the cervix a full bladder will displace the uterus backward, and a full rectum will displace the uterus forward.

Retrodisplacements of the uterus:

1. First degree retroversion: The fundus is on a level with the promontory of the sacrum and the cervix is in the midline axis of the vagina.

2. Second degree retroversion. The fundus is in the hollow of the sacrum and the cervix points toward the symphysis.

3. Third degree retroversion: The fundus is at the level of sacrococcygeal junction and the cervix points behind the symphysis.

Also, in retrodisplacement we can have some degree of retroflexion which would change the axis of the cervix to the vagina.

Table 1 shows the positions of the uterus in the last 300 patients seen at the Gynecological Clinic at Lafayette Charity Hospital. This includes both white and colored, both postpartal and gynecological patients.

TABLE 1

POSITION OF THE UTERUS

	No. Cases	Percentage
Acute Anteflexion	2	.6
Anteflexion	70	58
First Degree Retroversion	80	27
Second Degree Retroversion	34	11.4
Third Degree Retroversion	10	3
	300	100

Ninety per cent of patients with third degree retroversion have no symptoms whatsoever as a result of the retroversion. Of the remaining 10 per cent other pathology counts for the symptoms such as pelvic inflammatory disease, tumors, etc.

TABLE 2

CASE REPORTS

Race	Age	Marital Status	No. Children	Symptoms	Diagnosis	Operation
W. F.	17	Single	0	Dysmenorrhea	2nd degree retroverted uterus	Kelly-Simpson
W. F.	20	Single	0	Dysmenorrhea	Anteflexed, uterus 1st degree prolapsed	Kelly-Simpson
W. F.	42	Married	6	Pain in both sides	Chronic salpingitis; 2nd degree retroverted uterus	Kelly-Simpson
W. F.	36	Married	4	Pain in back and both sides, leukorrhea	Relaxed perineum lacerated cervix, 3rd degree retroverted uterus	Ant. & Post. Repair, D. & C. Cauterization of cervix, bilateral salpingectomy Kelly-Simpson suspension

Of these 10 cases in the Clinic of third degree retroversion all were treated with insertion of a pessary, and in half of them after wearing the pessary six weeks, the uterus was in anteflexion, and in the other half there was first degree retroversion and no symptoms.

TABLE 3

NUMBER SUSPENSIONS DONE AT LAFAYETTE CHARITY HOSPITAL FROM 1941-1948

1941	39	
1942	22	
1943	14	
1944	1	For teaching purpose only.
1945	0	
1946	1	For teaching purpose only.
1947	1	For teaching purpose only.
1948	0	
TOTAL	78	White, 71; Colored 7.

Ages: 17-42
77 Operations—Kelly Simpson
1 Operation—Baldy Webster

Table 2 shows not what to do but what you should not do. The doctor states that there are one hundred different types of operations for suspension of the uterus. This alone proves to me that there is no way to do a suspension as there is only one way to do anything right. Also, I notice that one of the cases in the doctor's series was done with one of the indications being dyspareunia. To me the difference between dyspareunia and libido is a question of salesmanship, and I am at a loss to understand how a suspension operation will relieve this complaint. Personally, I have never done a suspension operation. I have never seen a case in which I felt that the operation was indicated, even using the criteria that is given by the men who do the operation. I am inclined to place the operation in the category of the 75 per cent useless elective operations. I had not heard of the operation for the past ten years until I came to the State of Louisiana.

In closing, I would like to quote a personal communication from Dr. Carter who is Professor of Obstetrics and Gynecology at Duke University: "I will not allow any type of suspension done on any ward or private patient so long as I am in charge of Obstetrics and Gynecology at Duke."

STELLATE BLOCK: INDICATIONS AND A NEW APPROACH*

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NEW ORLEANS

Patients complaining of pain in the upper extremity are familiar to us all. The cause of this pain may be obvious, as in a recent fracture, or it may be obscure as in causalgia. Regardless of the etiology of the pain, relief for a variable period may nearly always be obtained by means of procaine block of the stellate ganglion on the affected side.

The exact reasons for obtaining this relief are not completely known, but one fact is made obvious by the results obtained, and that is that the autonomic nervous system is in some way directly concerned with the transmission of pain impulses, for by blocking the pathway through this system variable relief of pain is obtained.

We are all familiar with the division of the autonomic nervous system into the sympathetic or thoracolumbar and the parasympathetic or craniosacral sections.

*Read at meeting of the Orleans Parish Medical Society, December 8, 1947.

All peripheral nerves are accompanied by fibers of the sympathetic system. All impulses going over these fibers arise or terminate in the thoracic and lumbar segments of the central nervous system. The three cervical sympathetic ganglia all receive their impulses through the first thoracic sympathetic ganglion. When this first thoracic ganglion is fused to the inferior cervical ganglion the two together are referred to as the "stellate ganglion." By recent common usage the first thoracic sympathetic ganglion is generally referred to as the "stellate ganglion." Regardless of the name, it is this first thoracic sympathetic ganglion that is blocked in order to paralyze all sympathetic fibers going out over the brachial plexus.

In the past three years I have had occasion to utilize these blocks in over three hundred varied cases and as is true of all forms of treatment some conditions have been found to respond more favorably than others. The following conditions are those in which the response is such that the use of blocks should be given serious consideration.

Contusions and Crushing Injuries

The diagnosis in these cases is obvious. The pain, often severe is commonly treated by narcotics and it occasionally becomes difficult to wean patients from these drugs. The use of blocks will not only markedly decrease or totally abolish the pain felt but by also improving the blood supply will decrease swelling and promote healing.

Fractures and Dislocations—Post-Reduction Pain and Swelling

Painful swelling of the hand following fracture reduction and application of a cast commonly occurs. Treatment usually consists of elevation of the part, sedation, and sometimes splitting of the cast. If stellate blocks are performed on these patients the pain is usually completely relieved and the swelling markedly decreased. The same is true of dislocations that have been reduced and splinted. There is but one exception. The blocks will not compensate for a faulty cast. If pressure areas or constrictive bands are present do not expect the blocks to alter the plaster.

Subdeltoid and Subacromial Bursitis

These conditions are characterized by the old familiar group of calor, dolor, rubor and tumor about the shoulder. There is in addition limitation of shoulder motion caused by pain. Treatment usually consists of deep x-ray, or of local needling and novocaine infiltration, diathermy and exercise; for the most part with good effect in one to two weeks. The results obtained by means of stellate block in these cases is almost without exception so rapid and so complete that you will only believe it when you see it for yourselves. Repeat blocks are seldom required for these cases.

The Anginal Syndrome

Many patients experiencing repeated anginal attacks complain bitterly of the pain referred to their arms and shoulders. Because of pain the arm is often held close to the body and not used. Within a week these cases may develop a "frozen shoulder" and the pain and dysfunction continues but now from a different cause. This condition may be confirmed by noting the limitation of shoulder abduction and the almost complete absence of external rotation.

A number of physicians look upon anginal referred pain as a warning signal to the patient and are hesitant about methods of relief. In cases where the pain is intractable and the patient is developing a "frozen shoulder" something should be done. Stellate blocks will give them relief for which they will be grateful. What part the sympathetic cardiac nerves play in this I do not know, for they are also blocked by the procedure. The point is that the pain can be relieved without risking injury to the patient's cardiac status.

Diffuse Myositis

Patients experiencing pain along a muscle group, usually following exposure, an infectious process, or some form of muscle injury are ordinarily treated by sedation, diathermy, and physiotherapy. Relief may be accentuated by the use of blocks. The

results in this group are not as dramatic as in the other conditions mentioned.

The Scalenus Anticus Syndrome

Some authorities deny the existence of this syndrome and I wish that I could agree. There is no doubt but that some of the cases have been misdiagnosed but then there are others that closely fulfil the criteria. In these cases the scalene muscles may be felt as hard and fibrous, almost woody in consistency. Physiotherapy, which occasionally gives relief initially will in the long run not improve the condition. Blocks in these cases give but transitory relief and that is why they are important. If satisfactory relief is obtained the patient does not have the syndrome.

Posttraumatic Causalgia

These patients have often had a trivial blow to the region of the hand or wrist and little or no initial pain or trouble. Some days to weeks later they begin complaining of constant pain which may be severe enough to prevent use of the hand. Physical examination, x-rays and laboratory findings are uniformly negative. Relief in these cases is usually complete when blocks are performed. Repeat blocks may be necessary and if they are the patient usually asks for them.

The Frozen Shoulders

This condition has already been mentioned under the anginal syndrome. It more commonly results from the periarthritis following an acute subdeltoid or sobacromial bursitis or from injuries in the region of the shoulder. In these cases pain relief is good but repeated blocks are necessary. The increase in range of shoulder motion is superior in these cases when blocks are used over conventional methods of treatment. For best results the blocks should precede physiotherapy.

Technic:

There have been many technics devised for performing these blocks and they have commonly followed direct anterior, anterolateral or posterior approaches. All of these are a bit difficult and quite risky and a mortality rate has been attached to the procedure. There is, however, a simple lateral approach in which we do not try to directly infiltrate the area about the ganglion but rather infiltrate the region of the middle cervical ganglion. The ganglion chain is in a common sheath and by infil-

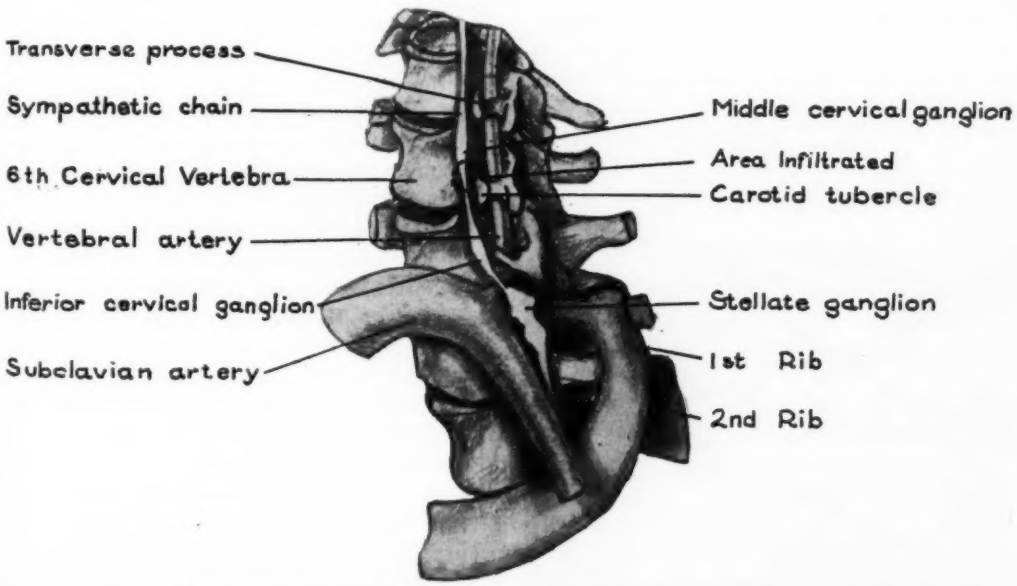

Figure 1. Skeletal relations of the sympathetic ganglion chain and the vertebral artery

trating into this sheath at a higher level and sitting the patient up we can anesthetize the stellate ganglion without putting a needle in close approximation to it.

Figure 1 shows the skeletal relations of the sympathetic ganglion chain and the vertebral artery. The carotid tubercle which is the anterior tip of the transverse process of the 6th cervical vertebra is our main landmark. This is palpable in the neck about 1½ inches above the junction of the inner and middle thirds of the clavicle on palpating through the anterior scalene muscle just beneath the clavicular head of the sterno-cleido-mastoid muscle. Our needle picks up this point and we work the tip medially along the transverse process for about 3/8th of an inch and then bring it anteriorly about 1/8th inch to get within the sympathetic sheath. The vulnerability of the vertebral artery when other approaches are used is apparent. The cupola of the lung is not shown on the diagram but it domes upwards from the inner edge of the first rib so that the stellate ganglion is in a groove between it and the neck of the rib. That is why pneumothoraces may be obtained when conventional anterior and antero-lateral approaches are used.

In performing the block the patient lies on his back with his head on a low pillow, his face turned away from the side being injected as in Fig. 2. The neck is palpated and the location of the tubercle of the 6th cervical transverse process determined. The area is then prepared and draped. A 10 cc. syringe is filled with 2 per cent novocaine and a skin wheal raised with a hypo needle. The needle is then changed to a 1¼ in. 24 gauge one. The index finger of the other hand is placed over the tubercle and pushed inwards and upwards, displacing the carotid sheath structures and making the tip of the tubercle practically a subcutaneous structure. This bony point is then picked up by the needle which is worked into position as previously described. The needle is then held in this position while the finger displacing the neck structures is released. The syringe is removed from the needle and the drop of fluid remaining in the hub watched while the patient inspires deeply. If a vein or artery has been entered blood will well out. If, by any quirk the spinal canal has been entered clear fluid will well out. If the needle is too low and the pleural space has been entered the fluid in the hub will be sucked in. In either case, discontinue the procedure. If all is well, re-attach the syringe and inject about 1 cc. Watch the patient for about one minute for any adverse reaction. If after this time conjunctival injection is noted you know that the needle is correctly placed and 3 to 4 cc. of the remaining novocaine is slowly injected. If the conjunctival injection is not noted, move the tip of the needle slightly, recheck as before and inject 1 cc. until the injection is noted. Immediately after completing the entire injection sit the patient up so that the novocaine will gravitate down the sheath and reach the stellate ganglion.

Figure 2. Site of injection in stellate block.

If a satisfactory block has been performed the first change noted will be the conjunctival injection followed by the appearance of Horner's syndrome: Ptosis, myosis, enophthalmos and anhydrosis. The hand of the blocked side should become quite warm and the patient will experience relief of pain.

Precautions:

As in all surgical procedures there are certain reactions and accidents that may occur. With proper care these may be minimized.

1. The patient should always be ques-

tioned for possible sensitivity reactions from preceding novocaine injections such as a tooth extraction.

2. Seconal grs. 1½ or one of the quick acting barbiturates should be given at least thirty minutes prior to the procedure to ward off novocaine reactions. If a reaction does occur intravenous barbiturates should be given immediately. Therefore intravenous sodium amytal and the means of giving it should be on hand before starting the procedure.

3. If a bloody tap is made or the pleural space or the spinal canal is entered the block should be postponed.

4. Never use over 25 cc. of 2 per cent novocaine in any block as this is the toxic limit of the drug. You will usually only require from 5 to 10 cc. Two per cent novocaine is better than 1 per cent as it more effectively penetrates the myelin covering of the nerves.

5. Never block both sides simultaneously. Occasionally the recurrent branch of the laryngeal nerve or the phrenic nerve is blocked. You would not want to do this bilaterally.

6. On the left side do not bring the needle in too horizontally or the esophagus may be punctured.

These various accidents should not happen if the technic given is closely followed. lowed.

There is one other reaction which you will be sure to see and that is the reaction to a needle. In these cases the patient complains of dizziness or feeling weak often before the procedure is done or any drug injected. This sort of performance is hard to distinguish from a mild reaction to the barbiturate. The only way is through knowing your patient.

In concluding, the following general prognosis as to results may be drawn. The more recent and acute the painful condition complained of, the more striking and complete will be the relief obtained through blocks. The more chronic the condition, the shorter will be the duration of pain relief, and repeated blocks will be necessary to obtain the final desired result.

AN UNUSUAL CASE OF A FOREIGN BODY IN THE ESOPHAGUS

J. R. HANLEY, M. D.
and
J. P. SWEARINGEN, M. D.
NEW ORLEANS

This is a case which is not at all infrequent, the swallowing of a coin with lodgment in the esophagus. Although this type of foreign body in the food passages is considered one of the commonest types, this case is atypical in the position assumed at the site of lodgment.

CASE REPORT

An 80 year old colored female was first seen in the accident room of Charity Hospital at 8:00 P.M. on January 3, 1948, complaining of some difficulty in swallowing and substernal discomfort. Her present illness began at 7:00 A.M. when she became aware of the fact that a 50 cent piece which she placed in her mouth the night before was missing. The patient then assumed she had swallowed this coin and made several attempts to cough it up without any success. After eating a light breakfast consisting of dry toast and coffee, the discomfort in her chest continued and she decided to come to the hospital. Prior to this there was no history of attacks of choking, gagging, dysphagia, aphagia, dyspnea, or involuntary coughing.

The review of other systems, family and social histories was non-contributory.

The past history revealed that the patient had a habit of sleeping with some kind of a coin in her mouth.

The patient was then admitted to the ward with a temperature of 98.4° F., pulse rate of 92; respiratory rate of 20; and blood pressure 190/100.

A cursory physical examination revealed only slight cardiac enlargement downward and to the left, with a prominent P.M.I. and a short systolic murmur in the aortic area. The urinalysis was normal. EPA view of the chest showed slight cardiac shadow enlargement and a disc-shaped opaque foreign body in the midline in the region of the aortic knob which was thought to be the coin which the patient swallowed. At this time additional films were ordered to include another EPA and a lateral view.

Since it was doubtful as to the exact location of the foreign body and the chest X-rays showed the edge of the coin erect, in the saggital or anterioposterior plane, bronchoscopy was considered primarily indicated. The patient was premedi-

Presented at meeting of the Eye, Ear, Nose, and Throat Staff, Charity Hospital of Louisiana, New Orleans, March 17, 1948.

cated with morphine sulfate gr. 1/8, atropine sulfate gr. 1/150, and sodium luminal grs. 2, and using 4 per cent cocaine topical anesthesia a bronchoscopy was performed. After a thorough examination of the trachea and both main stem bronchi, the bronchoscope was withdrawn and an esophagoscopy was performed. During the latter procedure the coin was located at the level of the aortic arch lying now in the coronal or transverse plane. It was removed with ease and the patient returned to the ward in excellent condition. The following morning, January 4, 1948, she was discharged from the hospital.

This case is unusual since the roentgenogram of the chest revealed the coin situated in the saggital or anteroposterior plane in the thoracic portion of the esophagus. It has long been taught that when a circular flat foreign body appears in the roentgenogram of the neck or chest, if its long axis stands in the anteroposterior position, it is probaby stuck between the vocal cords, since this position corresponds with the long axis of the glottis. If the long axis of the object stands in the coronal plane it must be in the esophagus, since the long axis of the esophageal diameter is in the coronal plane. It must be remembered that the esophagus is only a potential cylindric tube, compressed and flattened anteroposteriorly in the superior mediastinum, between the trachea in front and the bodies of the vertebrae behind. Actually the anterior and posterior walls of the esophagus are almost in complete apposition and are normally separated only by food or air.

Jackson and Jackson[1] explain the positions of this type of foreign body in the food or air passages in the following manner: "Coins and other disklike objects naturally lie in the frontal plane, because the shape and action of the tongue anteriorly combine with those of the posterior pharyngeal wall posteriorly to make the long diameter of the pharyngeal lumen transverse. On the other hand to enter the glottis, a flat object must lie in, or nearly in, the saggital plane. In other words, the muscular activities of the pharynx throw flat foreign bodies into the frontal plane, whereas they must be in the saggital plane to pass the glottis."

A case similar to this was reported by Schwartz[2] in which a patient swallowed a 25 cent piece. It was assumed that the coin passed through the entrance of the esophagus in the usual manner, but somehow passed down the extreme left of the esophagus and rotated into the anteroposterior position, perhaps through striking the bulge of the arch of the aorta. Thus the posterior edge of the coin became wedged in an esophageal pocket, beside the body of a vertebra, while the anterior edge was similarly wedged in a pocket in the left of the trachea, with the left stem bronchus preventing the coin from passing further down the esophagus. This interpretation was confirmed at the time of esophagoscopy.

REFERENCES

1. Jackson, C., and Jackson, D. L.: Diseases of Air and Food Passages of Foreign Body Origin. 1936.

2. Schwartz, Virgil J.: Unique case of foreign body in the esophagus, Arch. Otolaryng., 45:562, 1947.

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THE SPECIFIC TREATMENT OF FRACTURED ANKLE*

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NEW ORLEANS

Fractures involving the ankle are probably the most common type of fracture of the weight bearing joints. Surgeons caring for these injuries are familiar with the therapeutic difficulties. In some types satisfactory reduction is difficult. Some types while easy to reduce are difficult to retain, especially over the period of time necessary for consolidation. Some fractures result in nonunion. Early weight bearing is beneficial in some, and harmful in others. It it the aim of this presentation to suggest definite therapeutic indications in the management of these fractures.

FRACTURES OF EXTERNAL MALLEOLUS

Simple fractures of the external malleolus without widening of the ankle mortice require immobilization in a plaster cast extending from the base of the toes to below the knee for a period of four to six weeks. Weight bearing may be permitted immediately. If obvious tibiofibular diastasis is

*Read before the Sixty-eighth Annual Meeting of the Louisiana State Medical Society, Monroe, April 14, 1948.

associated with the fractured external malleolus, manual reduction is possible but prolonged retention by plaster of Paris is difficult. The diastasis tends to recur as the swelling subsides and the plaster becomes loose. This is especially prone to occur if early weight bearing is permitted. The treatment of choice is to insert a metallic transfixion screw through a small incision over the external malleolus that will firmly hold the tibia and fibula together in anatomical relationship. It is important that this be inserted with the foot held at a right angle so as not to interfere with subsequent dorsiflexion of the foot. The ankle is immobilized by a short leg plaster cast. Weight bearing is not permitted for at least three to four weeks in order to allow the ruptured inferior tibiofibular ligaments to heal. Immobilization is continued until union is evident by x-ray examination during which time weight bearing is encouraged. The screw as a rule does not require removal after consolidation has taken place.

In some cases the presence of tibiofibular diastasis is suggestive but not definite. It is advisable in this situation to repeat the x-ray examination under proper anesthesia with the surgeon manually attempting to displace the astragalus laterally on the fixed tibia.

Occasionally a fracture is encountered that involves the external malleolus and distal fibular shaft in which the proximal fragment becomes lodged behind the tibia. Manual reduction is impossible. Treatment consists of open reduction and reposition of the fibular fragments. The tibiofibular diastasis is then corrected by tibiofibular fixation with a transfixion screw.

FRACTURES OF MEDIAL MALLÆOLUS

Isolated fractures of the medial malleolus are not common. These fractures are usually associated with fractures of the external malleolus, and frequently with fractures involving the posterior articular surface of the distal end of the tibia. If no displacement is present, immobilization in a walking cast is indicated until x-ray evidence of union is present. If the fragment is displaced and if anatomic opposition cannot be obtained manually, open reduction is necessary. It will usually be found that a flap of fibrous tissue has become interposed between the fragments. This fibrous flap must be removed, the malleolus accurately replaced and fixed by a metallic screw to secure union. Displaced fractures involving the distal half of the medial malleolus should be excised to prevent nonunion.

FRACTURES OF BOTH MALLEOLI

Fractures involving the external and internal malleoli are generally caused by abduction or adduction strains. In abduction fractures the distal fragments together with the astragalus are displaced laterally and tibiofibular diastasis is present. Closed anatomic reduction of the abduction fracture is not always possible, and further it is difficult to maintain reduction in many instances. This fracture is best treated by open reduction and correction of the tibiofibular diastasis by a transfixion screw together with open reduction and internal fixation of the internal malleolus. The fracture can then be safely protected by a short leg plaster cast. Weight bearing should not be permitted for at least three to four weeks. Immobilization by a walking cast should then be continued until x-ray evidence of union is present.

In adduction fractures the distal fragment is displaced medially without tibiofibular diastasis. Closed reduction is usually possible and the fracture remains stable. Occasionally open reduction and internal fixation of the medial malleolus is necessary. Since tibiofibular diastasis is not a factor, weight bearing in a plaster cast may be started early and continued until union has occurred.

In fractures involving the external and internal melleoli without displacement, immobilization in a plaster of Paris cast below the knee is sufficient. Weight bearing may be permitted from the start and immobilization continued until there is x-ray evidence of union.

ANTERIOR AND POSTERIOR FRACTURES

Fractures involving the posterior articular surface of the tibia are common in

ankle injuries and are usually displaced. It is rarely seen as an isolated lesion. Fractures with displacement that involve less than one-third of the posterior articular surface of the tibia do not require reduction. If one-third or more of this surface is displaced, open reduction and internal fixation by a metallic screw inserted from a posterior approach is necessary to prevent posterior subluxation of the astragalus.

A type of ankle fracture involving the anterior half of the tibial articular surface and distal end of the tibial shaft is seen occasionally. It is usually due to falling forward and landing on the dorsiflexed foot. Considerable comminution of the fragments and anterior subluxation of the astragalus is usually present. The treatment of this injury is unsatisfactory. Closed reduction by traction and plantar flexion of the foot should be attempted. If unsuccessful and if comminution is not great, open reduction and internal fixation is indicated. Otherwise, the ankle joint should be arthrodesed primarily to avoid disabling traumatic arthritis.

SPRAINED ANKLE

The sprained ankle is a serious injury. In many instances the external lateral ligaments are completely ruptured. If this is not recognized and treated as a simple sprain, the ligaments fail to heal and recurrent subluxation of the ankle results. In every severe sprain the patient should be put under an anesthetic, either local or general, the foot forcibly maintained in inversion, and an A. P. x-ray taken. If the x-ray does not show tilting of the astragalus out of the mortice, the ligaments are not ruptured and treatment by any method is adequate. If tilting of the astragalus is evident by x-ray, the ligaments are ruptured, and the ankle must be immobilized in a plaster cast for a minimum of six weeks during which time weight bearing may be permitted.

SUMMARY

The treatment of the fractured ankle should be designed to restore and maintain anatomical relationships of the displaced fragments whenever present. This is best accomplished by open reduction and screw fixation especially when tibiofibular diastasis is present. Fractures without displacement or those with displacement but without diastasis can be adequately treated by closed methods. Recognition by special roentgenography of rupture of the lateral ligaments in sprained ankles is necessary and adequate treatment to prevent recurrent subluxation of the ankle is essential.

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A SURVEY OF THE STRUGGLE AGAINST STATE MEDICINE

The physicians of this nation now face the prospect of state taxation for universal medical care. The proposals for such a plan in previous years have taken the form of bills in Congress which were given a sympathetic reception by the administration, but were not a part of the party program. Now the situation is different.

The President has repeatedly stated his desire for such legislation and while it was not in the party platform it is in the legislative program of the leader of the party. It is understood the leader has what amounts to an obsession to see this program in law. We are intimately concerned to know what are the chances of such laws being enacted. It is clear that a party majority would be expected to support a party measure if the individual members expect any patronage. It is also clear that those members of Congress who supported the Dixiecrat ticket are more needed now by the administration then they were before the recent election. Many of these members of Congress from the South may have personal dislike for state medicine, but may have a desire to vote for it if they think a majority or even a vociferous minority of their constituents wants it.

In the national polls, which were uniformly wrong, about 7 to 11 per cent of those individuals interviewed were "undecided". It is now seen that these were the ones who determined the course of the election.

It must be recognized, however, that the Democratic party plurality has declined in each presidential election year since 1936, when it was seven mililon, to 1948 when it was only two million. Still more important is the fact that half the eligible voters did not vote at all.

Accordingly, we, as doctors, must not assume that the cause of freedom is lost and that each election pushes us reluctantly down "the road to serfdom". Actually the doctrine which says in effect let the strong carry the weak until they both drop is losing votes at each election. The well organized minority is still in control. The power of this administration, so far as medicine is concerned, could be weakened even at this hour if consistent opposition were directed against it. We have to help the undecided group to make up their minds and to tell the forty seven million citizens who did not vote, that American medicine is about to be undermined and that presently their liberties will be in even greater danger.

Such a program must be national, state and local. Up to the present the educational program has not been effective and national leadership has been unaggressive. We must reach the average voter. The state

societies can initiate such a program; the local societies can then give it expression. As a major part, the Auxiliary which has already been of great service should be further assisted.

In Louisiana we have a good start in dealing with the community aspects of medical care. Educational programs have been going on for several years. More recently the Council on Medical Serivce and Public Relations of the Louisiana State Medical Society has presented an excellent program calculated to reach those people who know little of the problem they face. Hospital and surgical insurance is becoming increasingly available. Our system of charity hospitals supplies assistance to a portion of the population that only taxation can provide for. We learn that as a result of these measures the representatives of our state in Congress have less clamor around their ears for state medicine than any other state group.

The most persuasive resource of all is yet to be utilized. This is the opportunity the physician has to explain the situation to his patients day by day. When each doctor discusses the problem with each patient over the whole nation within a year the undecided voter will know his interests and the sleeping citizen will have less lethargy. After this the men in Congress will know how to vote.

ORGANIZATION SECTION

The Executive Committee dedicates this section to the members of the Louisiana State Medical Society, feeling that a proper discussion of salient issues will contribute to the understanding and fortification of our Society.

An informed profession should be a wise one.

REPORTS TO EXECUTIVE COMMITTEE

INTERIM MEETING OF AMERICAN MEDICAL ASSOCIATION

The Third Interim Meeting of the American Medical Association was held in St. Louis on Tuesday, November 30, and Wednesday, December 1, 1948.

On the first day the House was called to order by Speaker Borzell at 10 a. m. sharp. The Reference Committee on Credentials reported 129 delegates certified at that time with more registering continuously.

The next order of business was selection of the recipient of the general practitioners' award. Twenty-three names, from twenty-three states, were submitted to the Board of Trustees, from which they selected three names for presentation to the House of Delegates. Louisiana was honored by having its representative, Dr. Charles M. Horton, among those presented to the House of Delegates, however in the voting Dr. W. L. "Buck" Pressly, of Due West, South Carolina, received the highest number of votes and was declared the recipient of this year's award.

During the address of the Speaker, the committees were appointed and Dr. James Q. Graves was appointed to the Blood Bank Committee and Dr. Val H. Fuchs was appointed to the Reference Committee on Amendments to the Constitution and By-Laws.

The first certificate and gold medal given to a layman by the AMA was awarded to Dean Alphonse M. Schwitalla, S. J., for his "services to the public welfare and American Medicine on a national level."

The rest of the morning session was spent in receiving committee reports until recess at 12:20 p. m. for lunch.

The afternoon session began at 2:00 o'clock and a number of resolutions were introduced until adjournment at 4:55 p. m.

Wednesday morning was spent in committee meetings. Amalgamation of the Blue Cross and Blue Shield into a national insurance plan was discussed in open meeting for three full hours during which time the committee members heard pros and

cons before turning in their report to the House for action.

The afternoon session began at 1:30 p. m. One hundred and seventy-three delegates were certified. After an executive session to discuss some problems, the Committee on Changes in the Constitution and By-Laws reported changes clarifying and simplifying executive sessions. The House now considers three types of sessions with the following differentiation: 1. Open sessions, when anyone can be present, including laymen and newspaper reporters. 2. Closed sessions, when laymen and newspaper reporters are barred. 3. Executive sessions, when only duly elected officers of the AMA and certified delegates will be permitted to attend. It is felt that this last type of session will be necessary only on rare occasions. This classification of sessions seems to be a very satisfactory settlement of a frequently occurring problem.

The Secretary reported that on October 1, 1948 the membership roll contained 139,779 names and the Fellowship roster was increased to 78,331. Increase in membership over the nine month period was 4,258 and that in Fellowship 2,053.

Protest that some hospitals are practicing medicine was answered by the committee as follows: "The analysis of the laws of the various states concerning what constitutes the practice of medicine has not yet been completed by the Bureau of Legal Medicine and Legislation of the Association. Until these laws are collected, studied and appraised by both the Bureau and the Committee on Hospitals and the Practice of Medicine, it seems absurd to attempt to make a report on the subject assigned to this Committee. However, certain observations are definitely in order. This report will be, or could be, used as a background for our thinking until the abovementioned information is available, and for action after there has been an opportunity to study the legal wording of the various state laws. It is assumed at the beginning that all of the laws will differ somewhat and that many things will be *legal*, not necessarily ethical, in one state and illegal in another. The interpretation of the various state laws will have to be studied in the light of judicial actions, if any, that have been made in the application of the laws to specific cases . . . Until the laws of all the states are available for study and until recommendations can be made that will hold water, both ethically and legally, it behooves us to attempt to settle at the local level all matters of controversy between medical men and the various institutions where they work and practice. This can be done if personalities can be forgotten and physicians are men of good will and will sit down around a table and find out, first, on what points agreements can be reached; second, what points exist where differences of opinion are slightly divergent; third, what points are of maximum disagreement, and finally, if it can be resolved to compromise everything but broad general principles concerning that which is for the public good. It will be for the good of every physician."

The Council on Industrial Health reaffirmed its previously expressed approval of joint planning for industrial health by management, labor and medicine.

Financial support from the Government for teaching in medical schools should be accepted only after all other sources are exhausted and only as a last resort, it was agreed.

Action at the last session in regard to pay for selective service examinations was rescinded.

A recommendation was adopted to revalue the education of medical students to stress general practice rather than the specialties and to advise a two-year internship in place of the present one year.

The government is asked in Selective Service to defer premedical and medical students until the completion of their studies, including not more than two years of internship.

The Council recommended to the Board of Trustees that they press legislation which will make the Surgeon Generals of the Army, Navy and Air Corps the advisor of all medical set-up instead of the laymen in charge of logistics; in addition, that the

chief medical officers in all branches of the Armed Services have complete control of all regulations in regard to health, hospitalization, evacuation and sanitation.

Objections were raised to the usage of Veterans Administration Hospitals except for service-connected disabilities and under indigent circumstances.

Objections were raised to the erection of so many Veterans Administration Hospitals. It was felt that hospitals should not be erected in localities where hospital facilities are already sufficient.

It was reported that compulsory attendance at staff meetings was never a requirement for AMA approval of hospitals.

In a resolution adopted unanimously, it was urged that state medical societies consider the introduction of legislation making rebates illegal in states where these are not already illegal. It was also urged that state societies receive complaints concerning rebates and hold hearings and make investigations at their own expense to eliminate the practice.

It was decided to increase the activities of the Washington office of the AMA and to employ additional personnel to carry out its increased work.

The Board of Trustees was authorized to assess the members of the AMA $25.00 this year in order to meet the added expenses to carry on activities during the coming year.

The proposal to form a national insurance company by the Blue Shield and Blue Cross for voluntary, prepaid medical and hospital care was not approved. As a substitute the House approved formation of a "national enrollment agency." This would coordinate all existing, approved medical and hospital care plans. It would also help to sell and distribute these services. Further development of coordination of reciprocity among the local plans were urged. It was also urged that in localities where plans are not in effect every effort be made to organize such plans at the earliest possible time.

The afternoon session adjourned at 5:55 p. m.

The meetings for the general practitioners were held at the same time and were very well attended. With two days yet to run the attendance at the close of Wednesday evening was 4,089. It was felt that by the close of the meeting the total attendance would reach the 5,000 mark.

In addition to the President, Dr. Hargrove, the Secretary, Dr. Talbot, and the Editor of the Journal, Dr. Jones, many other doctors from Louisiana were present at the meeting.

Dr. O. B. Owens and Mr. Frank Lais, of the Louisiana Physicians Service, and Drs. Guy Jones and J. P. Sanders, of the Committee on Rural Medical Service and the Academy of General Practice, attended a caucus of the Louisiana delegation during one of the luncheon intermissions.

Respectfully submitted,
Val H. Fuchs, M. D., Delegate
J. Q. Graves, M. D., Delegate.

Between the dates of November 27 and December 3, 1948, there was held in St. Louis one of the most important series of meetings ever sponsored by the AMA. On Saturday, which was known as Public Relations (PR) Day, there was presented a program of particular interest. This dealt with questions involving participation of lay groups and civilian workers in the ever-expanding field of public relations. So many of the large national lay organizations are far ahead in this field. The extent to which we are turning to such groups for inspiration and guidance was particularly noticeable. From the program one would deduce that our chief activity, from here on, should be to concentrate our efforts on the consumers of medical care (the public) and make them aware of the grave situation they would face if and when federalization of medicine became a reality. On the whole, the medical profession has been fairly well propagandized on these dangers through various mediums. We must get public opinion in our favor—this was the theme of all discussions, as I analyzed them. We need more talks to lay groups. Collateral agencies, in particular, must have a closer relationship with the

public and help to mold public opinion.

On Sunday, November 28 and Monday, November 29, I had the pleasure of attending the sessions of the Annual Conference of State Secretaries and Editors. The highlight of this program was the Symposium on Medical Legislation (National). Never has there been such frank and down-to-earth talks on the seriousness of the national situation, especially accentuated by the November 2 election. Dr. Edward J. McCormick of Toledo, Ohio, speaking as a member of the Board of Trustees of the AMA, presented a picture of the deplorable situation. His theme, forcibly delivered, was constructive criticism of the AMA for its complacency in this grave emergency. He emphasized that there is an emergency and that the time is rather short to do anything about it—but the situation is not hopeless. He read a letter, full of sarcasm, from a doctor in California, in which was put forth his own plan for the defeat of federalized medicine. This is as follows:

"1. Continue to sit on your fat derriere and do nothing.

"2. Be apathetic and, like 5,000,000 registered Republican voters who failed to vote, do not bother to make your opinion known. If you think, as they did, that your opinion or your vote is not worth anything, the opposition will agree with you and act accordingly, as they have.

"3 Write an occasional letter to your congressman, tell him off, and then explain proudly to the interns in the surgical dressing rooms how smart you are and what a stinker your congressman is.

"4. Tell everybody you see that the gag is up, and we might as well prepare for the inevitable.

"5. Moan and groan and issue explosive and unprintable epithets.

"6. Refer to your medical leadership as a group of impotent, ineffective and bumbling ignoramuses.

"7. Make speeches before sympathetic lay audiences, and convert those who already believe in free enterprise.

"8. Don't bother to tell your county society heads, your state society heads, or your national association heads what you want them to do. Expect them to find a way for you without your guidance.

"9. Scream about high medical society dues and forget that our friends in the trade unions demand many times what we pay; in other words, make the situation as difficult as possible, then grumble about it.

"10. Oppose any program developed by the majority of your colleagues because it demonstrates your superior wit and your general greatness.

"11. Remain superbly and learnedly dignified when Joe Doakes asks why you oppose state medicine. Brush him aside with any insult you can think of. Joe will like you for that.

"12. Don't bother to use the selling methods which actually bring messages before the public. Continue to depend on occasional radio feature programs. Billboard advertising, newspaper advertising, radio advertising and, above all, continuous and daily radio spot programs over the national hookups and all such like are too commercial, too troublesome, too expensive and too undignified; don't use them.

"13. Above all, disregard the 'little guy'—the one with a vote. Tell him nothing; push him around. He doesn't know anything anyway."

Dr. McCormick pleaded for the expansion of the Council on Medical Service in Washington by the AMA which, he feels, has heretofore only been an information bureau. He stressed that the AMA should not permit subsidiary or allied medical groups to express, publicly or otherwise, opinions on medical legislation without clearing them through the parent organization. He particularly censured the Academy of Pediatrics for putting forth in Washington opinions contrary to those of organized medicine and for its support of the contention that children are not getting adequate medical care because there are not suffi-

cient pediatricians when, as a matter of fact, 80 per cent of all children's diseases are treated by the general practitioner. Such a statement is fantastic.

Dr. McCormick answered the question, "What shall we do now to meet the emergency?" as follows:

1. Imminent and close cooperation between the AMA and state societies. This would provide uniformity of action. The AMA should inform the profession regarding its stand on any medical legislation which may be controversial.

2. Data should be sent from each state to the AMA concerning the present position of Congressmen on pending medical legislation.

3. The state medical society officers should be in constant touch with their Congressmen in Washington.

4. The Washington office of the Council on Medical Service of the AMA should inform the state societies of activities of their Congressmen which are contrary to the position taken by organized medicine.

5. Bulletins put out by the AMA should contain information concerning its position on current medical bills.

6. Conflicting opinions by state societies should be avoided.

7. The AMA should advise the public of its position on medical legislation of a controversial nature.

8. The AMA should be close advisor to its state societies.

9. The AMA should introduce legislation to counteract objectionable features in medical bills.

10. Only competent and well-informed physicians should testify before committees in regard to the position of the AMA.

11. The county medical societies should be bolder in their approach to controversial medical legislation and should keep in close touch with their representatives and senators.

12. At all times a constructive program should be developed both for the physicians and public.

13. With regard to speakers, it is recommended that public debates on controversial medical legislation should be avoided; especially appearances at forums over the radio. At times these cannot be avoided and in all instances such participants should be well informed on the subject matter presented.

14. Contact labor and other groups who may be opposed to our concepts and try to convert them to our position.

15. No county, state or collateral groups of the medical profession should state the position of organized medicine without conferring with the AMA.

16. Every doctor in the United States should be educated on the subject of federalized medicine and the job of opposing this legislation should not be left to the Board of Trustees of the AMA alone.

17. Editors of journals and other medical periodicals should not deprecate the AMA for reasons that they cannot justify. The place to settle our differences should be in the House of Delegates, committees, and conferences. Division and disunity weaken our structure.

Dr. Dwight H. Murray of Napa, California, discussing national medical legislation from the point of view of the state societies, brought out some very interesting facts. For example, in 16 states medical legislation has been introduced for the purpose of socializing medicine. None of the bills has been passed. He stated that the members of the medical profession should get into state politics as individuals but that the state societies should always remain neutral. He also stated that of the 140,000 doctors in the United States only 2,000 are working and carrying the load for the profession. Physicians also should be willing to make talks and such organizations as the Women's Auxiliary should be brought into the picture. And don't forget the legislator's wife. We now need members of the societies, willing to tackle this serious legislative program. The legislator's private physician, Doctor "X", might be the right man. After all, the fight will not be a very easy one. We must confer with allied groups and make friends with new forces, especially the insurance and banking people—they should be most cooperative.

All forms of publicity should be helpful and we must seek the aid of druggists, dentists, nurses, hospital administrators, etc. Get good team work. Do not only write letters after the damage has been done. Dr. Murray especially emphasized that all of this activity costs money and this certainly is no time to quibble about financial matters. Our opponents with their experts and billions of dolllars behind them are most active. The fight does not seem hopeless. We must send aid. Now is the time.

Representative Forest Harness from Indiana gave a very interesting talk on the serious situation that now exists in Washington and the firm ground on which the proponents of federalized medicine seem to be standing. Some of his thoughts were as follows: The present government is imbued with bureaucratic philosophies which they have nurtured for several years and which they are now planning to crystalize since their victory on November 2. The time has come for the medical profession to step out on the street corners, roll up their shirt sleeves and talk to the man on the street who does the voting. We should get away from academic trends and face things on a more practical basis. Now is the time to put this philosophy into action. Representative Harness recommends that we find a way to improve our public relations and that we make medical and hospital services available to all groups. The profession should assist in promoting ways and means for increasing the number of physicians, nurses and hospitals. Behind this is the serious implication against the medical profession that it is inactive and complacent. It is high time that something new be tried and developed in practical application. Mr. Harness stated there is nothing in the present picture to invite optimism. However, the program to fight federalization of medicine should be continued if we do not wish to lose individual liberties.

It was also my privilege to sit in on other programs. I was particularly interested in the Conference on Medical Society Radio Programs. At this conference was discussed present and future status of programs which have been on the air, sponsored by the AMA and some of the state societies. From all appearances, this seems to be a most effective means of conveying to the public the stand of physicians on various questions of comunity interest and, at the same time, acquainting them with the position of organized medicine in relation to federal legislation. It is felt that it will be some time before we can make use of television for this purpose because it is hard to get good script writers and artists for the presentation.

On Monday morning we had the distinct pleasure of listening to Dr. Paul R. Hawley who is the Chief Executive Officer for the Blue Shield-Blue Cross Commission. He reiterated with great emphasis his opinion that it will be necessary to combine the Blue Cross and Blue Shield plans in order to meet national demands of industrial concerns employing people in various states. He was very adamant in his position that unless this combination is carried out the federal government or some other agency will step in and undertake to satisfy the public.

In the above presentation of facts and opinions I have tried to present to you some of the constructive points and decisions reached at this all-important meeting. In doing this I feel that you should be properly informed concerning national trends in order that our State Society might mold and prepare our activities in line with the policies adopted by the AMA. I hope you will realize that we have a great responsibility in this regard and that we are facing it with sincerity and faith in our cause. There will unquestionably be presented to you a more detailed report by the House of Delegates of the AMA in respect to action taken on some of the important subjects spoken of above.

It was also my privilege to attend a meeting of the National Conferencec of County Medical Society officers where a most wonderful program was presented. Highlights of the program were presentations by Rear Admiral Joel T. Boone of the Navy, Brig. Gen. George E. Armstrong of the Army, a

representative from the Public Health Service, a representative of the Atomic Energy Commission and last, but not least, Dr. Maurice H. Friedman. The first four gentlemen spoke principally on the needs of the medical profession in the event of a national emergency in the near future. Also, they offered proposals for developing plans in municipal and damaged zones to meet situations which might develop as a result of atomic bombing. The last speaker, Dr. Friedman, presented a break-down of statistics accumulated by the government, incident to the rejection of draftees for physical defects during the last war. You know that these statistics prepared by the government have been erroneously used to make the public believe that if more medical care had been available such a large proportion of draftees would not have been rejected. An interesting feature of these government figures is that they do not include the volunteers but only those who were drafted, which made the percentage of rejections much higher. I am sure that you all have read in Louisiana papers, as well as medical journals, the testimony of Dr. Friedman last year before the Senate Committee on Labor and Welfare at which time he showed, without fear of contradiction, that the statistics presented by the government in this regard were absolutely fallacious in the majority.

I believe that you will agree with me that we are today facing one of the greatest tests to confront the medical profession. It is hoped that I have indicated some avenue of approach to the problems facing us in my attempt to bring to you a report of the recent meeting of the AMA.

Respectfully submitted,

P. T. Talbot, M. D.,
Secretary-Treasurer.

LOUISIANA STATE MEDICAL SOCIETY NEWS

CALENDAR

PARISH AND DISTRICT MEDICAL SOCIETY MEETINGS

Society	Date	Place
East Baton Rouge	Second Wednesday of every month	Baton Rouge
Morehouse	Second Tuesday of every month	Bastrop
Natchitoches	Second Tuesday of every month	
Orleans	Second Monday of every month	New Orleans
Ouachita	First Thursday of every month	Monroe
Rapides	First Monday of every month	Alexandria
Sabine	First Wednesday of every month	
Second District	Third Thursday of every month	
Shreveport	First Tuesday of every month	Shreveport
Vernon	First Thursday of every month	

TRIBUTE TO DR. MORGAN SMITH

Members of the Seventh District Medical Society:

Be it resolved that we here pay tribute to our professional friend and colleague, Dr. Morgan Smith, for his inestimable services and for his gracious consideration for his fellow workers; for the aid which he so willingly rendered in uplifting the mental, physical and social life of our community and for his valuable contributions to the field of medicine.

It was the happy lot of our late associate, Dr. Smith, to make for himself a fortunate life and to be given the satisfaction of knowing that the ample fruits of his labors were to remain for the enrichment of this community.

Be it further resolved that in recognition of the long and untiring services of Dr. Smith, we place a copy of this tribute in the minutes of the Seventh District Medical Society and that a copy be sent to the New Orleans Medical and Surgical Journal and another copy sent to Mrs. Hattie Smith in deep appreciation of true worth.

EAST AND WEST FELICIANA BI-PARISH SOCIETY

The Bi-Parish Medical Society of East and West Feliciana Parish met in the Educational Room of the Clinton Baptist Church.

After an excellent dinner prepared by Mrs. Willie B. Hubbs and aids, the Society met for a business meeting and scientific program.

Officers elected for 1949 are as follows: Dr. L.

S. Nolan, Jackson, Louisiana, President, Dr. Paul Jackson, Clinton, Louisiana, Vice President, Dr. E. M. Toler, Clinton, Louisiana, Secretary-Treasurer. Delegate to the Louisiana State Medical Society meeting, Dr. Glenn J. Smith, Jackson, Louisiana, Alternate, Dr. Paul Jackson, Clinton, Louisiana.

Scientific program was as follows: Dr. Cecil Lorio of Baton Rouge, Louisiana, gave an interesting and instructive lecture on Celiac Syndrome. Dr. Lorio's lecture was favorably received by all members present.

Dr. W. E. Barker, of Plaquemine, Louisiana, gave an interesting discussion on medical economics or "Medicine at the Cross Roads." Dr. Barker's talk stressed the danger of bills that may be presented in the 81st Congress of the United States dealing with state medicine.

A resolution by the Society was passed unanimously concurring in the viewpoint of Dr. Barker. It also requested the Bi-Parish Secretary to present a protest signed by all the members of the Society asking the Louisiana Senators and Congressmen to vote against any bill or bills detrimental to the members of the medical and surgical professions of our state. We have the best system of medicine and surgery in the world; therefore, we most vehemently protest and object to any change or meddling by Congress in the medical profession of the United States of America.

Dr. J. C. Rudd of Baton Rouge, Louisiana, Dr. Hodges of Jackson, Louisiana, and Dr. R. J. Field of Centreville, Mississippi were visiting guests.

The Society adjourned to meet in the East Louisiana State Hospital, Jackson, Louisiana, the first Wednesday in March, 1949, at 7:30 p. m.

L. S. U. MEDICAL ALUMNI ASSOCIATION

The New Orleans District of the L. S. U. Medical Alumni Association will hold its election meeting February 2 in the Stein Room, Jackson Brewing Company.

At this meeting nomination of officers for the state Alumni Association will be made.

The New Orleans District will be host at a cocktail party to be given May 6 for L. S. U. medical alumni attending the Louisiana State Medical Society meeting.

FIFTH DISTRICT MEDICAL SOCIETY

The annual meeting of the Fifth District Medical Society was held in Monroe, La., at the Virginia Hotel, Dr. H. S. Coon, President, presiding. An excellent and highly interesting program was enjoyed by the 45 members in attendance. Dr. Francis E. LeJeune discussed carcinoma of the larynx and showed slides and a most unusual motion picture on the subject. Dr. Roswell D. Johnson spoke on newer advances in the treatment of diseases of the blood-forming organs in children, bringing to light many phases of this highly complex subject.

At the dinner the following officers were elected: President, Dr. Carl L. Langford, Ruston; Secretary-Treasurer, Dr. F. E. McCarty, Monroe; Alternate to the Councilor, Dr. Tom Dekle, Jonesboro.

Dr. W. L. Bendel reported on the present status of the Louisiana Physicians Service.

The meeting was honored by the presence of the President of the State Society, Dr. M. D. Hargrove, who presented the latest information concerning the plight of the medical profession as regards socialized medicine. He exhorted each member to extend every effort to defeat this threat through personal contact with patients, meetings in public and donation of funds.

It was regretfully reported that the following members had died recently: Dr. C. P. Gray, Sr. and Dr. H. V. Collins, both of Monroe; Dr. V. E. Dudley of Lillie, La.; and Dr. R. Harlow of Jonesboro, La.

The meeting was then adjourned. Dr. Fred A. Marx expressed his thanks to the membership for their cooperation during his term as secretary.

"DETAIL MAN"

Personally, we like most "detail men." In case you haven't had contact with one recently, may we refresh your mind on the subject? Were we *Linnaeus*, we might describe him thus: Genus: Homo Sapiens; Habitat: Distribution almost universal, but becoming scarcer in Middle West due to the draft and lack of enforcement of game laws. (Most Dox think there is a perpetual open-season on these chaps.) Description: A hardy perennial. (Webster's definition of perennial: "continuing or enduring through the year or many years.") And, Boy, does he continue to endure a lot!

Further Description: This sub-order of Homo Sapiens not infrequently is married, and sires one or more little detailettes who depend upon the parent shrub for food and raiment. He has the customary complement of manual and pedal appendages; also, two ears, two eyes, two lungs and —believe it or not—a heart.

Usually he is a gentleman, which in itself is saying a lot. Obviously this rare specimen has an inexhaustible fund of patience, otherwise he would not be willing to cool his heels in your reception room for long periods of time, awaiting your willingness and readiness to see him for five minutes. He knows, of course, that in order to impress him with your importance, he will have to sit on his quadriceps in the outer sanctum until you get darn good and ready to admit him to your august presence.

He is a non-poisonous plant. Contact with him engenders no long and lingering ailment. He may be touched with impunity. (In fact, we have known instances where he was "touched" for sev-

eral dollars worth of valuable samples, simply for the asking.) He is odorless and tasteless, but is not, as we have implied, without feeling. He may be, at his worst, the rambler type of plant, in that he rambles on past the few minutes allotted to him, but still he cannot be classed with Rhus Toxicodendron or the Spiny Cactus.

Often he is addicted to tobacco, but unless you first light a cigarette, usually he will refrain from doing so while in your presence. He knows from long and bitter experience that while *he* is non-toxic, some Dox can be poisonous as toad stools—especially to "detail men."

So if you see one of these roving, self-abnegative, hard-working, patient and pleasant fellows beginning to take root in your reception room, for Heaven's sake have the girl bring him in before he becomes a permanent potted plant before your very eyes. Because all of you know how much easier it is to dispose of cut flowers than a jardiniere full of flowering hydrangeas.

But seriously, Fellows, let's give these boys a break. We are busy, of course, but not *too* busy to spare a few minutes of our time when it easily might be of mutual benefit. Ever hear of the Golden Rule? Think it over sometime; it will do you good.

—J. Phil. Edmundson, M. D.

Reprinted from the Jackson County Medical Society Weekly Bulletin

NATIONAL GASTROENTEROLOGICAL ASSOCIATION 1949 AWARD CONTEST

The National Gastroenterological Association again takes pleasure in announcing its Annual Cash Prize Award Contest for 1949. One hundred dollars and a Certificate of Merit will be given for the best unpublished contribution on Gastroenterology or allied subjects. Certificates will also be awarded those physicians whose contributions are deemed worthy.

Contestants residing in the United States must be members of the American Medical Association. Those residing in foreign countries must be members of a similar organization in their own country. The winning contribution will be selected by a board of impartial judges and the award is to be made at the Annual Convention Banquet of the National Gastroenterological Association in October of 1949.

Certificates awarded to other physicians will be mailed to them. The decision of the judges will be final. The Association reserves the exclusive right of publishing the winning contribution, and those receiving Certificates of Merit in its Official Publication, the Review of Gastroenterology.

All entries for the 1949 prize should be limited to 5,000 words, be typewritten in English, prepared in manuscript form, submitted in five copies accompanied by an entry letter, and must be received not later than April 1, 1949. Entries should be addressed to the National Gastroenterological Association, 1819 Broadway, New York 23, N. Y.

SOUTHWEST ALLERGY FORUM

The Southwest Allergy Forum will meet in El Paso, April 4th and 5th, 1949.

NEWS ITEM

Dr. Roy Carl Young, psychiatrist and neurologist, attended the meeting of the Southern Psychiatric Association in Dallas, Texas, on December 7th and 8th.

NEW OFFICERS OF COMPONENT SOCIETIES

The following officers have been elected by their respective parish societies to serve for 1949:

Allen Parish Medical Society
President—Dr. W. R. Hargrove, Oakdale
Vice-Pres.—Dr. Gurdon Buck, Kinder
Sec.-Treas.—Dr. R. E. Pace, Elizabeth
Delegate—Dr. Gurdon Buck, Kinder

Ascension Parish Medical Society
President—Dr. Gerald Gaudin, Gonzales
Vice-Pres.—Dr. Earl Schexnayder, Donaldsonville
Sec.-Treas.—Dr. Dawson T. Martin, Donaldsonville.
Delegate—Dr. P. H. LeBlanc, Donaldsonville
Alternate—Dr. H. A. Folse, Donaldsonville

Avoyelles Parish Medical Society
President—Dr. Filmore P. Bordelon, Marksville
Vice-Pres.—Dr. H. G. Temple, Bunkie
Sec.-Treas.—Dr. S. R. Abramson, Marksville
Delegate—Dr. A. M. Abramson, Marksville

Beauregard Parish Medical Society
President—Dr. Luke M. Marcello, DeRidder
Vice-Pres.—Dr. Sam T. Roberts, Sr., DeRidder
Sec.-Treas.—Dr. Thomas R. Sartor, DeRidder
Delegate—Dr. John D. Frazar, DeRidder
Alternate—Dr. Luke M. Marcello, DeRidder

Caddo Parish Medical Society
President—Dr. J. P. Sanders, Shreveport
1st Vice-Pres.—Dr. Keith Mason, Shreveport
2nd Vice-Pres.—Dr. Charles L. Black, Shreveport
Secretary—Dr. H. M. Yearwood, Shreveport
Treasurer—Dr. L. L. Davidge, Shreveport
Historian—Dr. A. A. Herold, Sr., Shreveport

Claiborne Parish Medical Society
President—Dr. J. E. Batchelor, Haynesville
Vice-Pres.—Dr. W. P. Gladney, Homer
Sec.-Treas.—Dr. M. L. Forcht, Jr., Haynesville
Delegate—Dr. Thomas M. Deas, Homer
Alternate—Dr. M. L. Forcht, Jr., Haynesville

DeSoto Parish Medical Society
President—Dr. G. Cooper, Mansfield
Vice-Pres.—Dr. H. P. Curtis, Mansfield
Sec.-Treas.—Dr. R. A. Tharp, Mansfield
Delegate—Dr. R. A. Tharp, Mansfield

Alternate—Dr. R. P. Thaxton, Mansfield

Jefferson Davis Parish Medical Society
President—Dr. G. McClure, Welsh
Vice-Pres.—Dr. G. G. Richard, Lake Arthur
Sec.-Treas.—Dr. L. E. Shirley, Jennings
Delegate—Dr. John G. McClure, Welsh

Rapides Parish Medical Society
President—Dr. Richard E. C. Miller, Alexandria
1st Vice-Pres.—Dr. Edward Claude Uhrich, Alexandria
2nd Vice-Pres.—Dr. Morris J. Hair, Lecompte
Sec.-Treas.—Dr. Harry Gahagan, Alexandria
Delegate—Dr. H. H. Hardy, Alexandria
Alternate—Dr. B. M. Wilson, Alexandria

Sabine Parish Medical Society
President—Dr. L. H. Murdock, Zwolle
Vice-Pres.—Dr. G. F. Weber, Many
Sec.-Treas.—Dr. A. A. Flores, Pleasant Hill

St. Tammany Parish Medical Society
President—Dr. Foote R. Singleton, Slidell
Vice-Pres.—Dr. Jos. F. Polk, Slidell
Sec.-Treas.—Dr. Durwood J. Thobodaux, Slidell
Delegate—Dr. Roy C. Young, Covington
Alternate—Dr. T. J. Healy, Covington

Webster Parish Medical Society
President—Dr. Joseph M. Garrett, Cotton Valley
Vice-Pres.—Dr. C. S. Sentell, Minden
Sec.-Treas.—Dr. C. M. Baker, Minden
Delegate—Dr. C. S. Sentell, Minden
Alternate—Dr. C. M. Baker, Minden

HERMAN VASCOE COLLINS
1882-1948

Dr. Herman Vascoe Collins, member of the Ouachita Parish Medical Society, died in Monroe on December 2, 1948. Dr. Collins was a graduate of the Atlanta School of Medicine in 1912. He was an active and interested member of the State Society since 1925 and was in practice in Monroe from 1938.

JOHN T. CREBBIN
1873-1948

Dr. John T. Crebbin of New Orleans died on October 20, 1948. He was a former member of the Caddo Parish Medical Society and later of the Orleans Parish Society. He was a graduate of the 1900 Class of the Hahnemann Medical College, Chicago, Ill.

LEO NEHEMIAH ELSON
1884-1948

Dr. Leo Nehemiah Elson of New Orleans died on December 12, 1948. He was a member of the Orleans Parsih Medical Society. Dr. Elson was a graduate of Tulane Medical School, Class of 1914.

COURTLAND PRENTICE GRAY
1882-1948

The Ouachita Parish Medical Society has reported the death of Dr. Courtland Prentice Gray of Monroe, La., in December, 1948. Dr. Gray was a member of the State Society since 1914, served as President 1935-1936 and actively participated in its activities. He graduated from the Baltimore Medical College in 1904.

EDMOND KLAMKE
1875-1948

Dr. Edmond Klamke of Alexandria, La., died on October 13, 1948. He was associated with the Rapides Parish Health Unit and was an active member of the Rapides Parish Medical Society, serving as second vice-president in 1945. Dr. Klamke was a graduate of the University of Copenhagen in 1901.

LEWIS HENRY PIRKLE
1873-1948

Dr. Lewis Henry Pirkle of Shreveport, La., died on November 5, 1948. Dr. Pirkle was a member of the Caddo Parish Medical Society since 1914. He was a graduate of Tulane Medical School in 1904.

BOOK REVIEWS

A History of the Heart and the Circulation: By Frederick A. Willius, M. D., M. S. in Med., and Thomas J. Dry, M. A., M. D., Ch. B., M. S. in Med., Philadelphia and London, W. B. Saunders Company, 1948, Pp. 435. Illus. price, $8.00.

This book is divided into three parts. The first part of two hundred sixty-five pages contains eight chapters. Each chapter is followed by a summary with adequate references. This part covers the period from antiquity to the first quarter of the twentieth century. An account is given of the work and contributions of those who have given most in building the foundation on which our present day knowledge now stands. The influence of different races is given. No less interesting is the influence of certain individuals on the progress of medical knowledge. The most ramarkable is that of Galen, whose influence swayed medical thought for fourteen and a half centuries.

The second part is composed of special biographies. In these ninety-five pages an account is given of the lives and works of those great personalities and observers who have contributed so much, each one from his own field of endeavor. It begins with Hippocrates (460-377 B. C.) ending with Sir Thomas Lewis (1881-1945). The story of these men, now as always, should be a source of inspiration. There are many quotations, as in the first part of the book that are worthy of consider-

ation even in our day, especially those of Sir James Mackenzie.

The third part of seventy-one pages is a chronological presentation of data according to subjects, arranged in alphabetical order. The first subject mentioned in this part of the book is anatomy of the heart and circulation. The first name mentioned under this subject is Imhotep, a somewhat mythical individual who was supposed to have lived about 3,000 B.C. In these pages one can quickly find the names of those who have made the most important contributions, when they lived, and what they did, by looking under the subject one is interested in at that moment—let it be anatomy, aneurysm, electrocardiography, or therapy.

The print and reproductions are good and the book is easy to read. Anyone interested in cardiovascular disease or any part of medicine will find it a quick source of information. The authors have rendered a valuable service.

J. M. BAMBER, M. D.

Venous Thrombosis and Pulmonary Embolism: By Harold Neuhof, M. D., New York, Grune & Stratton, 1948. Pp. 159. Price, $4.50.

This monograph is a report on extensive studies made by the author, acting on an assignment by the Medical Board of the Mount Sinai Hospital to study the role of pulmonary embolism as a cause of death and to work out a plan for its surgical management.

The frequent insidious development of venous thrombosis in the lower extremities and the difficulty of early recognition of its occurrence is stressed both in general statements and in the numerous illustrative case reports.

Surgical treatment of various thrombosis, as well as an evaluation of the present status of anticoagulant therapy, is presented in a critical manner.

The author fully discusses peripheral pulmonary embolism and also gives a detailed report on massive pulmonary embolism based in part on a study of 88 fatal cases.

Methods of treatment of massive pulmonary embolism are reviewed and the several reasons for an unfavorable attitude toward pulmonary embolectomy are cited. Although autopsy revealed that from topographical features alone, embolectomy would have been feasible in 33 of the 88 cases, there were amongst these 33 cases no distinctive clinical features to differentiate them from less favorable cases, thus leading to the inevitable conclusion that an operation always must be regarded as an exploratory one. Of the 88 cases almost half were suffering from otherwise irremediable conditions. There were between 15 and 26 cases over a ten year period in which favorable criteria for embolectomy existed.

The occurrence of death after the onset of sympsoms was within ten minutes in 18 cases; in eleven to fifteen minutes in 9 cases; in sixteen to thirty minutes in 11 cases; and in one half to one hour in 8 cases. Twelve died in one to four hours; 6 in four to ten hours; 5 in ten to twenty-four hours. From these observations the author concludes that a significant fact concerning the cases to be regarded as favorable for embolectimy is that the duration of life was short in many. As stated by the author, this is quite out of line with the protracted duration of life for which the Trendelenburg operation has been advocated.

Altogether, this volume is a most worthwhile publication, bringing together in monograph form a great fund of data as well as the author's personal thoughts concerning venous thrombosis and pulmonary embolism.

AMBROSE H. STORCK, M. D.

Pre-Operative and Post-Operative Care of Surgical Patients: By Hugh C. Ilgenfritz, St. Louis, C. V. Mosby Co. 1948. Pp. 898. Illus. Price, $10.00.

This well written book covers the difficult subject of preoperative and postoperative care of surgical patients in an admirable manner.

Starting with a discussion of fluid and electrolyte balance the author continues with a discussion of metabolism and nutrition, sedatives, general preoperative measures, shock, transfusions, organic diseases, chemotherapy, postoperative complications, and the preoperative and postoperative care of patiens with disease of various organs. There is also a good resumé of preoperative and postoperative treatment of wounds and burns.

A valuable feature of the book is the presentation of the physiologic basis for the therapeutic measures advocated.

The book reflects the author's extensive knowledge of the current literature and contains numerous references.

This volume should be of special value to surgical residents and interns.

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PUBLICATIONS RECEIVED

J. B. Lippincott Company, Philadelphia: New and Nonofficial Remedies—1948, issued under the direction and supervision of the Council on Pharmacy and Chemistry of the American Medical Association.

G. P. Putnam's Sons, New York: Anesthesia: Principles and Practicec, by Alice M. Hunt, R. N.

Charles C. Thomas, Springfield, Ill.: The Renal Origin of Hypertension, by Harry Goldblatt, M. D. Experimental Immunochemistry, by Elvin A. Kabat, Ph. D. and Manfred M. Mayer, Ph. D.

The Williams and Wilkins Company, Baltimore: Hygiene (Third Edition), by J. R. Currie, M. A., M. D., D. P. H., F. R. C. P. and A. G. Mearns, M. D., B. Sc., D. P. H., F. R. S.